THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. XXI FEBRUARY, 1929 No. 2

ACUTE MASSIVE COLLAPSE (ATELECTASIS) OF THE LUNG*

BY DAVID R. BOWEN, M.D.

Radiologist to the Pennsylvania Hospital

PHILADELPHIA, PENNSYLVANIA

DEFINITION

ACUTE massive collapse (atelectasis) of the lung is a definite clinical entity characterized by more or less airlessness of a lobule, a partial or entire lobe, a whole lung or even of both lungs. It is rarely seen without some measure of increased or retained secretion which may be excessive and which rapidly becomes purulent. Dependent upon a variety of predisposing factors, its final incidence is, practically without exception, due to complete occlusion of a bronchus or a bronchial branch with subsequent absorption of the retained air, and its area is definitely limited to that part of the lung served by the bronchus so occluded.

The most important premonitory symptom is a moderate elevation of temperature; the most notable subjective sign is dyspnea, and the most reliable physical and roentgen finding is displacement of the heart toward the involved lung.

Described with surprisingly few errors more than seventy-five years ago, it has in recent years been rediscovered as a postoperative complication. One object of this paper is to demonstrate that such a limitation of the subject is misleading and that the consideration of massive collapse may not be, with safety, omitted from any branch of lung diagnosis.

NOMENCLATURE

There is still a confusion of names which is a hindrance to study and perhaps even to understanding. Two terms, collapse and atelectasis, are in common use. Each word has the merit of correct derivation, common usage and clarity of meaning.

Analysis of thirty-eight titles of articles written since January 1, 1924, titles in which one or the other of these words was used, shows that collapse appeared twenty-three times and atelectasis fifteen times. The Quarterly Cumulative Index, published by the American Medical Association, lists both terms under the head collapse, there being no separate heading for atelectasis. Therefore, although my title was listed on this program as "atelectasis," with collapse in parenthesis, I have finally chosen to use the word collapse, placing atelectasis second to it. The stated objections are that the term collapse was previously applied to the condition of the lungs as found in pneumothorax and in pleural effusion. The fact is, however, that in both instances we think and write pneumothorax and effusion, collapse being a constant but unwritten part of the idea conveyed. While both words are absolutely correct, collapse is simpler and has the merit of distinctly wider use. I believe that broader

* Read at the Second International Congress of Radiology, Stockholm, Sweden, July 23–27, 1928, and at the Twenty-ninth Annual Meeting, AMERICAN ROENTGEN RAY SOCIETY, Kansas City, Mo., Sept. 25–28, 1928.

understanding of the subject will be furthered by using the term collapse exclusively. "Obstructive," used by Coryllos and Birnbaum,[41] conveys a thought which is very desirable for correct diagnosis and therapy.

"Apneumatosis," suggested by Fuchs[61] and by Hewitt,[84,85] and proposed again recently by Coryllos and Birnbaum,[41] has no greater merit of clarity and is, at best, an awkward word. In one title "non-pneumatic collapse" was used.

"Postoperative" is a widely employed and very useful adjective, even if it does lend distinction to a causative factor which is not always operative in this condition.

"Lobar" and "lobular" may be used as defining location and extent but should not be understood as distinguishing types.

"Active collapse" is, I believe, an inaccurate term which may very well be dropped.

HISTORY

The rediscovery of massive collapse of the lung and particularly the relating of this condition to postoperative and post-traumatic states dates from the 1910 paper of Pasteur;[159] but we find that, however its antecedents may differ, and whatever its surrounding circumstances may be, there is, in its pathology at any rate, nothing really new. With almost unbelievable lapses, its history goes back from that date for sixty years or more to contributions of extraordinary merit, and the more so if we consider the handicaps of that day.

Willshire,[219] writing in 1853, gives a detailed account of the events, beginning, he thinks, with Schenk in 1811, through which the differential diagnosis between lobar and lobular pneumonia had passed. He found evidence that "Wilbrandt, in 1816, Eberhard in 1817 and Lucae in 1819" followed, with clear recognition of a post-mortem state in the lungs of infants which they regarded as congenital or "due to the musculi bronchiales not being as yet sufficiently accustomed to the rhythmic order of respiration."

In 1830 Alderson,[2] writing on the "Pathology of Hooping Cough" describes 4 autopsies in children dying from that disease and gives an accurate description of partial collapse which he clearly distinguishes from any inflammatory process but to which he assigns no name. Willshire, however, credits him with first linking the atelectasis of Joerg with the assumptory collapse of the lung, afterward (as we shall presently find) playing so important a part in the hands of Bailly and Legendre.

Dugés in 1821 and Shallgruber in 1823 also gave clear descriptions of autopsy findings. Up to this time, however, Willshire says that all held the theory in one way or another of congenital non-expansion.

In 1834 Joerg[112] wrote on a particular condition of the lungs of newborn children and more fully worked out this subject in 1835, at which time, so Willshire believes, was first applied the term atelectasis. Barlow[9] in 1841 reported atelectasis from the pressure of dilated heart. In one case this seems to have been accentuated by obstructive bronchitis.

Gairdner,[63] in a review (1854) credits Dugeś (1821), Louis (1829) and Joerg (1832) with recognizing the condition post mortem, each of them calling it "carnification," and each realizing its points of difference from pneumonia. Louis found it in 19 out of 46 autopsies after death from typhoid fever. These were amplified in his work on fever in 1841. Louis offers no speculation as to the origin of the condition.

Rilliet and Barthez[172] in 1841 give this description of carnification: "The lung in this state is externally collapsed, soft and flaccid instead of full hard and resistant as in pneumonia. Its color is violet marked by white lines, disposed in lozenges or squares, defining the lobules. There is no crepitation on pressure. On incision, red color, smooth, resisting pressure of finger, so as to be penetrable with difficulty; exuding upon pressure a serus bloody liquid destitute of air. Its appearance, very like the close compact fibres of a muscle, has given it its name. The carnification may be marginal or some part of a lobe. The middle lobe is the only one we have seen entirely invaded." (Perhaps the first mention of a massive collapse.) They considered this condition as a terminal phase of pneumonia.

Willshire describes the ensuing con

troversy, which seems to have been heated and complicated by a scramble for priority. He concludes that "nothing less than a complete unsettlement of the whole subject as related to new born children and infants seemed inevitable, in consequence of the views thus being enforced concerning atelectasis." A sort of compromise, however, was attempted by some: it was admitted that a "fetal condition" of the lungs frequently occurred in children who died a short time after birth, and in whom the acts of respiration had been very imperfectly performed, and that, of course, mere atelectatic symptoms and lesions were not those of inflammation. Some at the same time sought to establish that if, under such circumstances, life had been prolonged to some extent, an inflammatory condition supervened, and then after death the effects of the two conditions were so intermixed as to baffle all solution. As to "carnification," some of its analogies to the atelectatic state were admitted, one or two regarded it as a result of chronic pneumonia, while others denied the explanations thus offered but could not afford any solution of the matter themselves.

We have thus stated enough to carry the subject onward to the year 1844 when the memoir of Legendre and Bailly appeared which caused quite a revolution in the history of lobular pneumonia.

Legendre and Bailly[123] established the autopsy identity of congenital non-expansion with a return to a similar state, "etat foetal," in lungs which had once expanded. Though they attributed the collapse to the elasticity of the lung parenchyma, acting because of lack of air entrance, their several articles created a profound impression and mark a distinct turn and advance in the literature. (Quoted under "Pathology" q.v.)

Mendelssohn [150] in 1845 and Traube[205] in 1846, in very similar research, experimentally occluded bronchi with shot, paper and (Mendelssohn) gum arabic paste, and produced collapse. Traube especially expressed doubt as to the mechanism and its practical import, and though he expressed the belief that "we stand here, as it is seen, on the confines of a territory that can only be conquered for science by sufficient clinical and pathologico-anatomical investigation, combined with systematically ordered experiments," he seems not to have entered much further into this "territory." Lichtheim, later, wrote of Traube as having marked the path for him to follow.

The first systematic treatise that I find originating in the English language is the first edition of Charles West's "Lectures on the Diseases of Infancy and Childhood" (1850).[216] (This book eventually ran through six editions, each of which was promptly re-printed in America.) In a chapter entitled "Collapse of lung that has once been expanded- described as lobular pneumonia by various writers—its characters, symptoms and differences from true pneumonia," West shows familiarity with collapse as a feature of disease of the lung and clearly recognizes its separation from the inflammatory states. Despite his belief that Legendre and Bailly were wrong in their conception of the etiology of atelectasis (they believed it to come on from engorgement of the pulmonary circulation), West says that "they have thrown more light on the affections of the lungs in infancy and childhood than all the writers of the previous ten years taken together."

As one goes only a little into the history of this matter, there develops the thought that each important contribution is promptly minimized by some word or act of the contributor. Thus West closes this lecture by excusing the brevity of it, "in consideration of its extreme rarity in this country."

Fuchs[60] in 1845 wrote in criticism of the then accepted conception of "lobular pneumonia" and considered it to be a disease condition of the mucous membrane of the bronchi combined with a contingent lesion of the pulmonary vesicles similar to that existing normally in the fetus, and proposed the name "apneumatosis" (see Nomenclature). Thus we see that the three terms proposed today, atelectasis, collapse and apneumatosis, were all used before 1850. Our choice need not be prejudiced by any thought that one or the other has important priority.

In 1850 Gairdner,[62] then a youth of

twenty-six and pathologist in the University of Edinburgh, wrote an extended study of lung collapse quoted at length in this paper. Very much therein stands unimproved to this day. His theory that collapse came from the action of partially inspissated mucus acting as a ball valve at bronchial bifurcations, is almost wholly discredited today, but has had from time to time worthy support, as by Dixon and Brodie, Jackson and others; and even now, can we be so sure that in the earlier stages of collapse this may not be to some extent a factor? Three years later Gairdner[63] wrote a painstaking review of the current literature of collapse in his time, and then for some reason, perhaps his growing enthusiasm for public health work, dropped it entirely. Though he continued a brilliant professional life with much writing to his credit, I am unable to find that he ever again wrote any word about lung collapse.

In 1852 Meigs[149] addressed the Philadelphia Medical Association on "Atelectasis Pulmonum." Confining his address to children, Meigs gave, however, a clear differentiation between this condition and bronchial pneumonia. Although West's book was republished in Philadelphia in 1850 Meigs seems to have drawn his inspiration not from West but from "a small work on Atelectasis Pulmonum,* by Dr. George A. Rees." Again, "it (the breathing) presented also the character presented by Dr. Rees of London, which is a peculiar sinking inwards, or depression of the walls of the chest during *inspiration*."

In a paper in the first volume of the Transactions of the New York Academy of Medicine, Foster[59] showed familiarity with the work of Alderson, West and Gairdner. He gave as causes: "Whatever occasions imperfect action of the muscles of respiration and whatever impedes the passage of air through the bronchial tubes." "These are causes which may operate at all periods of life." Under "treatment," Foster outlined a sensible nursing regimen with an occasional emetic, concluding: "If by these means we do not save our patients we shall at least have the satisfaction of knowing that we have not hastened their death by treating antiphlogistically maladies which have nothing in common with inflammation."

Stokes[200] (1837) understood mediastinal displacement as a sign and result of "vesicular emphysema of Laennec." Stokes' book must have been an authority in its day for it is more than worth while to read it now. With this in mind, it is difficult to understand how Gairdner, Jenner, Hewitt, Lichtheim, and others, before Pasteur, failed to recognize mediastinal displacement in the condition which may, for analogy, be termed the compensatory emphysema of massive collapse.

While the work of West, Meigs and Foster seems to represent the entire American knowledge at this time, there was much writing and counterwriting in Europe by those who did or did not agree with Legendre and Bailly, with Rilliet and Barthez[173] taking a middle ground. Hewitt[84] wrote in 1857 of apneumatosis, preferring this term taken from Fuchs to the more commonly used atelectasis. Jenner[111a] (1857) wrote: "While complete obstruction of a bronchial tube is followed by collapse of the pulmonary tissue on its distal side; partial closure of a tube has as its consequence, dilatation of some of the air cells of the part of the lung from which it leads, etc." This observation was confirmed fifty years later by the research of MacCallum. In 1861 and again in 1867 Bartels[11,12] made extended autopsy studies in the lung affections of croupous diphtheria, finding atelectasis as a complication when the bronchi were closed by the membrane. (Jackson and his colleagues have recently drained such cases by the bronchoscope.) Bartels accepted Gairdner's ball-valve theory but questioned whether complete collapse could come about in this way alone. In 1871 Hewitt furnished a chapter on apneumatosis for Reynolds' System of Medicine and Hertz[82] wrote of atelectasis in Ziemssen's Cyclopedia. It must be said, however, that with the exception of the work of Mendelssohn, Traube and Gairdner, atelectasis failed to attract general attention

* Gairdner also refers to this book as of 1850 but I have been unable to trace it further. Dr. Geo. Owen Rees wrote at this period upon a variety of topics, listed in the Catalogue of the Surgeon General's Library, but "Atelectasis Pulmonum" is not among them.

and that when noticed at all it was considered as an affliction of children.

In 1879 Lichtheim[126] repeated Traube's experiments more accurately by the use of laminaria tents. Combining bronchial occlusion with ligation of bronchial vessels, he proved conclusively that collapse would occur only when the blood circulation was unimpeded. Lichtheim also studied the rate of absorption of gases and found that oxygen or carbon dioxide was absorbed rapidly while the absorption of nitrogen was much slower. This has since been abundantly confirmed. This remarkable research by Lichtheim seems not to have been given any clinical application.

With the exceptions mentioned, nothing was added to the knowledge of atelectasis as the reviews of Gairdner and of Willshire left it in 1853. Sewall pertinently refers to this period as a "conspiracy of silence" and such it was so far as clinical application was concerned.

In 1890, Pasteur[157] published his paper on respiratory paralysis after diphtheria as a cause of pulmonary complications. Pasteur was impressed with the belief that the collapse found at autopsy, usually at both bases, was directly due to phrenic paralysis.

Cabot[29] in 1905 noted that "in most individuals a normal atelectasis of lung margins can be demonstrated by examination before and after forcible repeated respiration. Resonance may be increased over a width of one inch." (It does not appear that in this or any later edition was true massive collapse with heart displacement recognized.) Weinberger[214] in 1906 reported a left lung collapse from the pressure of an aneurysm upon the left bronchus. Barr[10] reported in 1907 what is possibly the earliest record of a postoperative lung collapse. It does not seem to have occurred to him that there was any definite etiologic relation proceeding from the operation—removal of tuberculous glands from the groin of a youth. Though the collapse was of a size to be primarily mistaken for pleural effusion, displacement of the heart was not recognized.

The first recorded recognition of heart displacement in a case of lung collapse after Stokes[200] seems to be that reported by Samuel West,[217] in 1907, in a case of plugging of the bronchus in plastic bronchitis. West sketched the displaced heart and mediastinum and studied its probable mechanism, arriving at the conclusion now generally accepted.

In 1908 Pasteur's second paper[158] appeared. Other cases of post-diphtheritic phrenic palsy were included and several autopsied cases of adults, in all of whom phrenic disability was a prominent feature. West's case was quoted but no further comment upon the heart displacement was made. This paper closed with the following sentence: "This observation may throw some light on the nature of post-operative lung conditions."

Also in 1908 MacCallum[141] published researches which see under "Etiology."

In 1910 Pasteur[159] wrote under the title "Active Lobar Collapse of the Lung after Abdominal Operations," and here the real modern conception of this condition began. At this time Pasteur makes the first recorded mention of heart displacement as a cardinal diagnostic sign. Also this is the first definite mention of collapse as a type of postoperative complication. Pasteur here predicts that "active collapse of the lung from deficiency of inspiratory power, will be found to occupy an important position among the determining causes of postoperative lung complications." In 1911 Pasteur[160] delivered the annual oration before the Medical Society of London: "Post-operative Lung Complications." Here was presented an analysis of 3,559 surgical operations in Middlesex Hospital, 1906–1910. There were 201 pulmonary complications with 45 deaths; 88 cases were classed as pneumonia and 12 as massive collapse.*

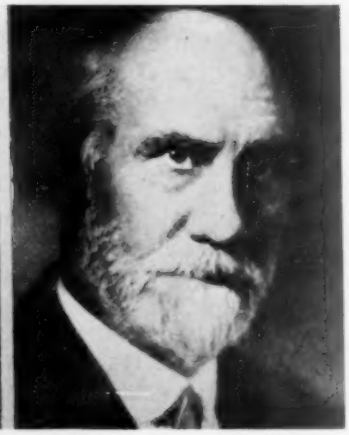
Pasteur's final contribution[161] appeared in 1914, and in point of description and diagnosis but little has been added. The practical stimulus given by Pasteur's work can scarcely be over-estimated,

* Comparing this 1911 oration with the paper of 1910 one discovers a curious illustration of the difficulty continually encountered in teaching the principles of massive lung collapse to those who are at the beginning. In 1911 Pasteur says: "The mechanism of the cardiac displacement is not difficult to understand," and in two lucid sentences he explains it. The year before, he wrote: "The heart signs require somewhat fuller consideration. The displacement toward the affected side is very difficult to account for." Then, following a labored quarter column he adds: "The explanation of this must, I think, be left open for the present." In that year (1910–11) Pasteur graduated from student to authority

though there has been and is a regrettable lack of familiarity with it. In American literature the paper usually quoted, perhaps because in American print, is the one of 1890. It seems that the condition dealt with in 1890 and 1908 differs widely from that so completely depicted in 1910–1911, and 1914. These continued quotations from the earlier papers and neglect of the later ones is no small factor in the difficulty still experienced in getting the truth to the top (see Types under Pathology). Also in 1914 Elliott and Dingley[53] reported 11 cases and confessed to a belief that many others had passed unrecognized. As a result of these observations and personal research, they definitely concluded that phrenic paralysis does not of itself cause massive collapse which they found due to bronchial obstruction. They rejected the theory of bronchial contraction or spasm. Pasteur's last contribution to the subject was a letter to the editor of *Lancet*[162] in which he took issue with these conclusions. Never wholly satisfied with "the reflex diaphragm inhibition" hypothesis, Pasteur was never able to accept the bronchial occlusion theory.*

Sir William T. Gairdner.

Dr. William Pasteur.



Dr. Henry Sewall.

This extended reference to Pasteur is given here, not only because he introduced a new era, nor wholly from the fact that he was first to recognize the great importance of collapse among postoperative lung complications, and first to point to heart displacement as the great clinical sign, but also because there is, as yet, no better way to gather a practical understanding of the subject than to study Pasteur's development of it in this unique observation over a period of twenty-four years.

During the World War, British surgeons, notably Bradford,[21] and Crymble,[43] reported numerous cases among the wounded and much speculation arose from the curious fact that many of these showed collapse contralateral to the injury. As to thought of etiology Pasteur seems to have been followed generally, particularly so by Bradford and by Briscoe,[24] who in 1920 published an elaborate study of the musculature of the thorax.

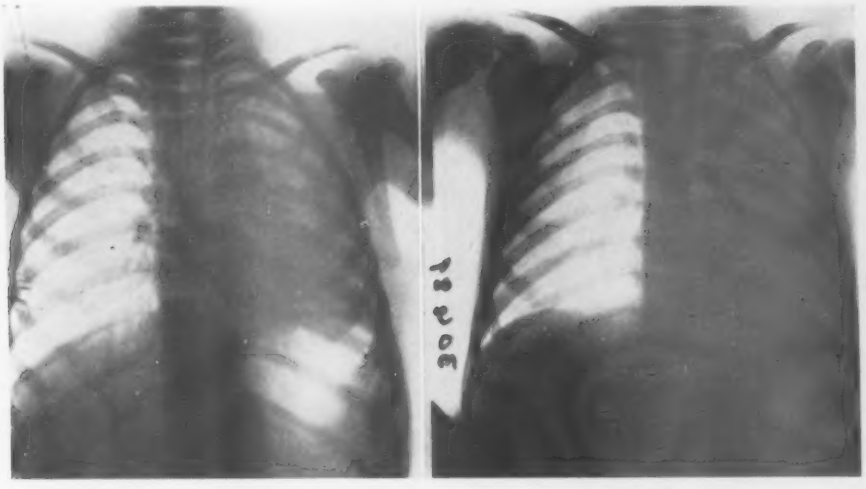
The first communication in American literature, after Pasteur, was the paper by Scrimger[188] of Montreal in 1921. Reporting 7 cases in a series of 540 operations, Scrimger tentatively held the theory of vagus reflex followed by viscid secretion and, finally, absorption of the retained air. In the same year (1921) appeared the very important work of Sewall[191] of Denver. Taking no notice of surgical or military reports, Sewall began his study with the works of Gairdner, Willshire and Lichtheim and developed his study as related to physical examination of the chest and

* In a recent (October, 1928) communication, Pasteur, now in retirement outside London, writes: "I am quite unrepentant as regards the plug theory which in my experience is only infinitely rarely the cause of lobar collapse, the condition I have described. I have come across only one well authenticated case, which was published by West of St. Bartholomew's. Having started the ball rolling I am naturally much interested as a pure spectator in what is happening to it."

particularly with regard to pulmonary tuberculosis. In this field Sewall's contribution stands almost alone. His repeated slogan, "Think Atelectasis," in other words, approach no chest examination without the thought that here may be ing directly with collapse, while in 1927 there were half that number in the one year.

ALLIED CONDITIONS

Under this heading are considered the following conditions:



B A

FIG. 1. Typical "drowned lung" from inorganic foreign body (small electric lamp). A—Before removal entire lung gives homogeneous shadow. B—Immediately after removal drainage is sufficient to disclose heart shadow and show bronchi as negative shadows, but heart displacement is greater than before because drainage has lessened the size of the affected lung and therefore increased emphysema of the opposite lung.

some appearance or phase of atelectasis, placed a much needed emphasis upon the frequency of non-traumatic massive collapse.

The invention of the bronchoscope by Killian in 1897 and its improvement by Jackson in 1904, and since, and the wide variety of conditions to which he has adapted it, began, even before Pasteur's work, to broaden the general understanding of intrapulmonary conditions of which very little had been known. This and Jackson's success as a teacher are certain to have an influence upon the subject in hand, the far-reaching value of which affords wide range for imagination.

The more recent history is well known, and its makers are liberally quoted elsewhere in this paper. As illustrative of the premise that massive collapse is everywhere waiting to be recognized, it is interesting to note the rapid increase in the number of essays upon this topic. In the six years, 1921–1926, there are in the accompanying bibliography 48 papers deal-

(1) those which are pre-existing and co-existing disease,

(2) those which differential diagnosis must rule out;

(3) those which occur as complications, and

(4) those which remain as sequelae.

Tuberculosis (Sewall[190]) is frequently complicated by large or small areas of collapse. Malignancy may produce collapse either by bronchial obstruction or by pressure upon lung parenchyma.

Diphtheria, either as diphtheritic bronchitis (Jackson[104,105]), or by its sequel, diphtheritic phrenic paralysis (Pasteur[157]) should never be handled without full consideration of collapse as a complication or sequel.

The complication, "drowned lung," a term first used by Johnston in connection with foreign bodies and referred to by Jackson,[100] Manges,[135] Leopold,[125] and others, exists as a complication in many cases. In fact, the writer has seen but one case report in which roentgen-ray findings

were recorded, where drowned lung can be definitely and wholly excluded (Fig. 1).

When we remember that the terminal bronchi are less than 0.5 mm. in diameter and that the alveoli are slightly larger, remembering at the same time the constant

related to collapse either as an antecedent, a complication or as equel, there is abundant evidence. Coryllos and Birnbaum suggest an even more important relation. In their study of experimental atelectasis in dogs[42] these writers were impressed with

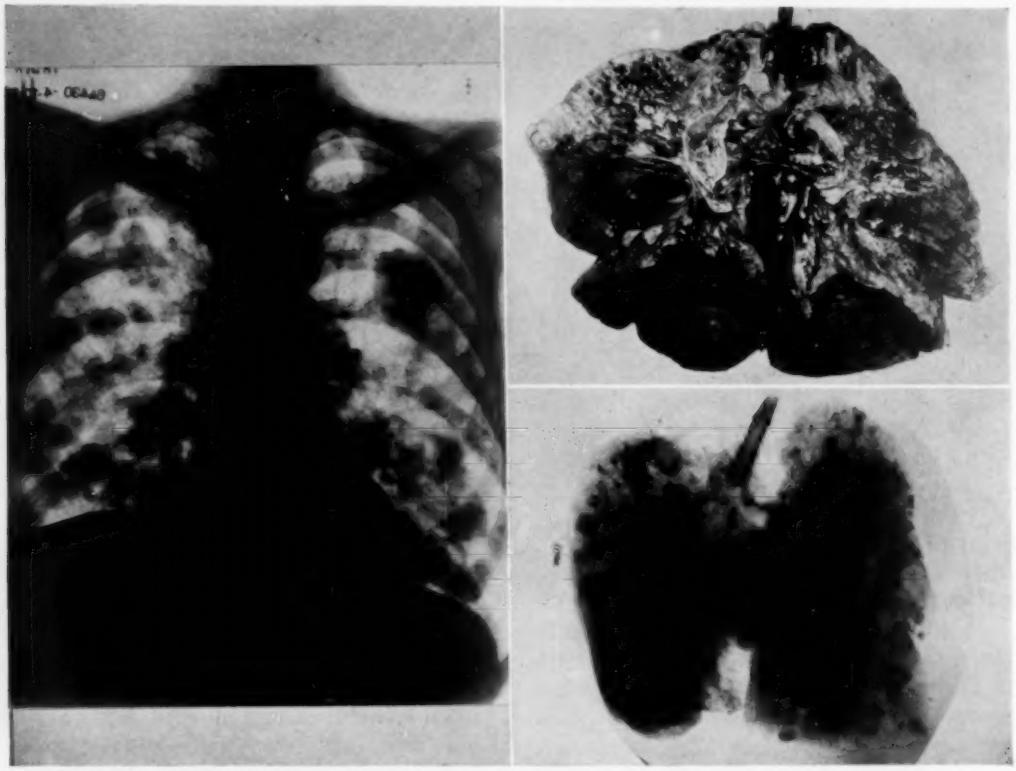


FIG. 2. Metastatic hypernephroma, shown here because of the extreme obstructive emphysema.

secretion of moisture needed as an adjuvant to oxygenization and for regulation of body heat, the absence of drowning is more difficult to explain than its presence. That it is, unquestionably, greater and more frequent in foreign body cases than in other forms of massive collapse is due to the fact that the obstruction occurs in these cases abruptly and without previous disturbance of lung secretion which continues until impeded by its own increasing pressure; while in massive collapse, as a complication, the great, and possibly only, determining factor is an inflamed, irritated or compressed mucous surface, the secretion of which has already slowed up or has ceased and become thickened by evaporation.

That pneumonia may be frequently

many points of similarity between this condition and lobar pneumonia. The more important which they have enumerated are as follows:

"(1) The lobar distribution.
"(2) The rapid onset of symptoms.
"(3) The almost identical clinical symptoms except for the most toxic element in pneumonia.
"(4) The very rapid improvement after the release of the obstruction in atelectasis, and after the crisis in pneumonia.
"(5) The more usual location in the inferior lobes, particularly the right.
"(6) The *x*-ray findings.
"(7) The gross pathology."

After developing their experimental work Coryllos and Birnbaum conclude that "lobar pneumonia is a pneumococcic massive atelectasis of the lung." . . . "Bronchoscopic liberation of the occluded bronchus has already given conclusive results in massive atelectasis

and even in post-diphtheritic pneumonia. If we are right in the interpretation of the clinical facts presented, bronchoscopic aspiration in pneumonia, at least in the early stages, would be indicated."

"A human case has been bronchoscoped and the findings corroborated our experimental observations. The bronchus corresponding to the affected lobe, and only this bronchus, was occluded by mucous exudate.

While this one case was perhaps not a brilliant one, it can hardly be doubted that the patient was benefited. Crisis started within four days, the chest was clear by physical and roentgen signs in nine days and the patient was discharged, cured on the fourteenth day. Be this as it may, careful observers can hardly doubt that, at least as a complication, collapse plays a very important rôle in many cases of lobar pneumonia.

Thoenes,[202] Wallgren,[210] and Griffith[70] have all written concerning displacement of the heart in the pneumonias of children. Thoenes regards these as due to complicating atelectases, while Wallgren and Griffith believe that the heart is displaced away from the emphysematous but otherwise uninvolved lung. The case reports all strongly suggest complicating, or perhaps pre-existing, collapse. It may be well at this point to go back and consider the dictum of Gairdner that" emphysema can only occur in the presence of atelectasis or collapse, which in some measure diminishes the volume of the other lung."

Bronchopneumonia, lobular pneumonia (if fairly stated, the pneumonia which is not lobar pneumonia) was discountenanced as a name by Louis, Legendre and Bailly, West, Williams, Gairdner, Fuchs, Bartels, Hewitt and Lichtheim, and still it is as constantly in use as if they had never lived and worked. May we not sum up the whole matter, in a manner dogmatic but at least brief, by accepting this forgotten dictum that: "Bronchopneumonia is a bronchitis with partial lobular 'patchy' collapse," the collapsed areas being infected with organisms less virulent than the pneumococcus? Certainly no other theory so readily fulfills the conditions. Lee and Tucker[119] quite accept this view that, "Arrest in these collapsed areas of the small emboli which are usually scattered through the circulation after all operative procedures involving blood vessels, as proven by Cutler, is an explanation of the mechanism of post-operative bronchopneumonia. The clinical picture of the so-called cases of post-operative or post-anesthetic pneumonia in so unlike true lobar pneumonia seen by the internist that it is strange that we have so completely accepted this diagnosis up to the present time."

Embolism and infarct have a relation to massive collapse which, at best, is obscure. Churchill says, "While pulmonary embolism does not cause atelectasis, it is possible that an area of collapse may alter the pathological course of an embolism lodging there." I believe that accurate diagnosis will surely reduce the number of cases of embolism and infarct as proportioned to massive collapse.

Abscess. The definite relation between lung abscess and partial lung collapse was well understood by Gairdner. He wrote:

"In regard to the characters of these abscesses, as distinguished from tubercle, my observations almost exactly concur with those of Dr. West. In one point alone I am disposed to differ from him. He seems to regard the abscesses as not formed in truly carnified lung, because the condensed portions are not always exactly circumscribed by the interlobular tissue. I believe this appearance will be fully accounted for by the fact that when bronchial abscesses occur, the bronchitis is usually general and intense, and the collapse correspondingly diffused. Besides, I have found bronchial abscesses, and their results, in exactly circumscribed lobules, in repeated instances; and that the tissue in which they occur is mostly collapsed there can be no doubt, from the whole of its ordinary and microscopic characters."

Churchill and Holmes[35] present 6 cases of chronic pulmonary suppuration showing lobar atelectasis. "This association may be explained either as atelectasis secondary to a focus of pulmonary suppuration or, as we suggest, chronic intrabronchial stagnation superimposed on a primary atelectasis. Such primary lobar atelectasis is found in the so-called massive collapse of the lung and 4 of our sick patients date their symptoms from inci-

dents known to be associated with this disorder." It is, of course, by no means implied that this is the only origin of non-tuberculous lung abscess. Such is certainly not the case.

Obstructive emphysema described first by Iglauer[97,98] and later elaborated by Manges and others of the Jackson Bronchoscopic Clinic, seems to be satisfactorily established by them as dependent on valve action by the obstructing body or substance, air entering more easily than it can depart. Jackson reports having seen this action taking place while watching a foreign body in the roentgenoscope. MacCallum[141] introduced sections of rubber tubing into excised lungs which were then suspended in a bottle so as to be expanded and contracted by changes in the negative or positive air pressure about them, and demonstrated that though alternately expanded and contracted, there was a continuous gradual increase of inflation. There are, therefore, two plausible theories of slightly differing mechanism either of which, in a given case, may have been the active process.

ETIOLOGY

A great part of the discussion of etiology is still on a theoretical basis: First, there have been put forward the causes external to the lung:

(*A*) Paralysis of the diaphragm or a part of it.

(*B*) Fixation of the diaphragm, because of pleuritis or by traumatism to the upper abdomen.

(*C*) Reflex action from injury or "insult" to the vagus.

(*D*) Various nerve injuries or disturbances causing incoördination of the diaphragm or parts of it with the other respiratory muscles.

Second, causes within the lung causing obstruction either from

(1) Partial plug acting as a ball valve, the air finally leaving the lung because, in these instances, the expiratory air stream is less impeded than the inspiratory.

(2) Actual plugging of the lung with collapse, caused specifically by circulatory absorption of the air distal to the obstruction.

To these are added many contributory causes.

Paralysis. Legendre and Bailly[123] hinted at this as a contributing cause in infants, judging that a partial cause was the weakness of the patient whereby air failed to reach parts of the lung, especially the thinner margins. The paralysis theory is mainly due to Pasteur.[157] His early work was with the postdiphtheritic phrenic palsies of children, and he found atelectasis, usually bilateral, in many autopsies. When, some years later, he began to study lung collapse as a postoperative complication, this long painstaking experience influenced his opinion mainly.

This theory was supported by Bradford[21,22] and others, but while Pasteur was doubtless right in ascribing to paralysis the major rôle in the postdiphtheritic cases it must be remembered that these were fatally ill children and that phrenic palsy in comparatively healthy adults has not been found to be a considerable etiologic factor. Elliott and Dingley[53] quite failed to so produce collapse experimentally. Sante[177] rejects this theory and, as do others, calls attention to the fact that it does not occur when phrenic palsy is deliberately produced in man as a part of the pneumothorax treatment of pulmonary tuberculosis. "Besides," he says, "you do not get paralysis of the diaphragm (in clinical atelectasis). It is immobilization in a high position, evidently a defense reaction. This high position is maintained after the lung is completely reinflated." Crymble[43] reports a war-wound case in which there was complete paralysis of all the muscles of respiration except the left half of the diaphragm without any opacity in the lung areas.

Fixation as a cause has been advanced by many. The specific mechanism within the lung would be similar to that of paralysis, the collapse occurring gradually because of lessened respiratory movement. Tidy and Phillips,[204] and Crymble[43] present this theory. Lee and Tucker[119] and many others accept it as a contributing cause, and we will consider it under that heading.

Reflex action, especially some interference with the vagus, has many supporters.

Bradford,[21] while mainly following Pasteur, suggests that the vagal stimulation of the constrictor muscles of the bronchioles may be a cause, but offers no evidence. Sante[177] quotes Bradford with approval and advances the theory that "once approximated, the walls of these bronchioles are held in apposition by cohesion, collapse rapidly following owing to absorption of the alveolar air by the circulating blood." Dixon and Brodie[48] lend some experimental support to this theory. They say that "the broncho-constrictor fibres run in the vagus; none are found in the sympathetic. Contraction of the bronchioles may lead to collapse or over-distention of the lung depending upon the force of inflation on one hand and the time allowed for deflation on the other. In the dog and rabbit typical bronchiolar constriction follows immediately vagal stimulation." On the other hand, Schroeder and Green[184] found that "repeated pinching of the phrenic nerve during operation (done deliberately for research) caused reflex response but no later symptoms." Later when, during removal of a tumor from the neck, they found it necessary to cut the phrenic nerve it was done unhesitatingly and resutured without untoward effect. Scott[185] believes that "we are justified in concluding that there is some reflex mechanism which produces, either directly or secondarily by secretion, an obstruction." There are many who accept this reflex as a contributing cause.

There remains the theory of Briscoe[24] that by various causes as, for instance, localized diaphragmatic pleurisy, a failure of coördination of respiratory muscles, particularly opposing portions of the diaphragm, the collapse results thereform as in the theories of paralysis and fixation. This would be quite easily dismissed were it not for the elaborately painstaking anatomical study that Briscoe has made of the respiratory musculature. Nevertheless, the conclusion seems to be unwarranted as a major factor in the production of collapse.

Auer and Lewis[7] conclude from an experimental study of anaphylaxis that: "Lungs of a guinea pig killed by immediate anaphylaxis are distended and in an inspiratory position so that the diaphragm is pushed down."

"This immobilization of the lungs, which is due to broncho-constriction, is of peripheral origin, for the destruction of the spinal cord and medulla affect in no appreciable way the promptness and extent of the response to the injection of the toxic dose." Whether this observation has any practical relation to our topic may well be doubted but those who are reluctant to drop the reflex theory of etiology may find the reference convenient. The production by MacCallum[141] of emphysema by broncho-constriction is referred to again.

Fig. 3. Illustration taken from Gairdner's 1850 thesis, showing his conception of the origin and valve-like action of the mucous plug.

Turning to causes within the lung, there are few writers who fail to note the presence of obstructing mucus in at least some cases. Pasteur never accepted this theory and Bradford rejects it as a major or frequent cause because of repeated failure to find such plugs at autopsy. Sante and others admit its presence but consider it as only a secondary cause.

The principal controversy among those who recognize the presence of a mucous plug as a main factor is as to the mechanism of its action. Legendre and Bailly[123] wrote of abundant, thick mucus which accumulates in the bronchial tubes of the infant, which at best expectorates only imperfectly, and add that "this mucus is positively the best obstacle for the free circulation of air in the bronchioles." But

it was Gairdner[62] who, in 1850, first undertook to ascribe to the mucous plug the major rôle in the production of collapse (Fig. 3).

"The bronchi are a series of gradually diminishing cylinders, dividing, for the most part, dichotomously. If a plug of any kind, but especially one closely adapted to the form of the tube, and possessing considerable tenacity, be lodged in any portion of such a cylinder, it will move with much more difficulty toward the smaller end, and in doing so will close up the tapering tube much more tightly against the passage of air than when moved in the opposite direction into a wider space. If such a plug be placed over a bifurcation it will, even if freely moving in the larger space in which it lies, be of sufficient bulk to fall back upon one or other of the subdivisions during inspiration, in the manner of a *ball-valve* upon the orifice of a syringe, and thus completely to occlude it (see Fig. 3). The consequence of this mechanical arrangement must inevitably be, that at every expiration a portion of air will be expelled which, in inspiration, is not restored, partly owing to the comparative weakness of the inspiratory force, and in part to the valvular action of the plug. If cough supervene, the plug may be entirely dislodged from its position, or expectorated, the air of course returning freely into the obstructed part; but if the expiratory force is only sufficient slightly to displace the plug, so as to allow of the outward passage of air, the inspiration will again bring it back to its former position, and the repetition of this process must, after a time, end in perfect collapse of the portion of lung usually fed with air by the obstructed bronchus."

Gairdner was impressed with the idea that he could actually hear this valve action taking place.

"When the tubes become really obstructed, the sound of vesicular respiration is at some points of the lung much diminished, or it may be altogether lost, and the bronchial râles are correspondingly modified, indicating the passage of a smaller quantity of air with a much greater amount of resistance; the ordinary mucous rattles being supplanted in part by the sharp 'clicking' and valve-like sounds so well described by Dr. Williams."

It is doubtful whether anyone today will accept this statement. Indeed, it seems that hardly any writer today is aware of the extent of Gairdner's really remarkable work, marred only by this one mistake. He went so far as to write with some derision of those before him who had suggested, theoretically, the possibility that circulatory absorption might partially account for atelectasis.

"It is not a little surprising that this simple and clear mechanical mode of explaining the collapse should not have occurred to Dr. Fuchs who, in accounting for the disappearance of the air in the experiments of Mendelssohn and Traube, finds himself reduced to the theory of its absorption into the blood-vessels. Surely nothing can be more superfluous than such an explanation."

The ball-valve theory was, I think, original with Gairdner, and his lucid presentation of it seems to have been accepted without question where his work made any impression at all, for the following thirty years, and was given unqualified support by Rilliet and Barthez.[172] It is true that Bartels[11] expressed doubt that the bronchi leading to the atelectatic areas always contained obstructing mucus; and expounded his own theory that the obstruction arose by spastic contraction of the bronchi from irritation, with a simultaneous swelling of catarrhal membrane. He argued that increased resistance causes a decreased inflation at inspiration while the expulsion of the air is brought about by compression to which the lung parts are subjected by frequent and violent coughing spells. This explanation evidently did not satisfy Bartels himself for he continues, "It does not seem plausible to me that this pressure from without upon the lung should alone be sufficient to completely deflate the lung."

It was Lichtheim[126] who first disputed, effectively, this theory, unfortunately magnifying Gairdner's error and minimizing the other truly remarkable aspects of his work. Lichtheim's great contribution was that of calling attention to his conviction that "all forms of atelectasis come about by absorption of the living air through the blood circulation." He proved conclusively that areas of lung tissue distal to a plugged bronchus became atelectatic if the circulation were not interfered with, but that no collapse occurred if the blood vessels to that part were tied. There seems little

purpose in calling attention to later expositions of the valve theory in the etiology of collapse, for in recent years it has been quite discarded. On the other hand, the conception of obstructive emphysema wholly apart from the theory of valve-like etiology is difficult, perhaps impossible. When, however, we remember that the autopsied lung never completely collapses I cannot imagine that valve-like action could possibly empty air from a lung, in life, against a constantly increasing negative intrapleural pressure—pressure great enough, as we shall see, to sharply displace the entire mediastinal structure and to continuously hold it to the unnatural position. Even if we must reject Gairdner's[62] ball-valve theory of the mechanism of collapse, he must be given credit for much. He knew what produced collapse if he did miss the mechanism of it. The following from his thesis might well have been written yesterday:

"In considering, as a whole, the causes which tend to produce bronchitic collapse, they seem to resolve themselves into the following:—Firstly, the existence of mucous in the bronchi, which is more liable to produce obstruction according as it is tenacious and viscid: secondly, weakness or inefficiency of the inspiratory power, however caused; thirdly, inability to cough and expectorate, and thus to remove the obstructing mucous. Of these conditions the first must be considered as the immediate exciting cause, the others as predisposing causes, co-operating with the first, but incapable without it of producing collapse."

Elliott and Dingley[53] are credited with the rediscovery of the mucus plug complex and, from a clinical standpoint, the credit seems due them. They studied 11 cases and confess that "undoubtedly more occurred during that time for we did not systematically study every case of post-operative pneumonia." They considered the possibility of collapse occurring (A) by active compression: (1) from outside, (2) the elasticity of the alveolar wall, (3) a hypothetical quasi muscular contractile power of the alveolar wall, and (B) by absorption of the air when the bronchioles are obstructed, owing to (1) inflammatory edema and secretion, (2) spasmodic contraction of the bronchiole muscles, and selected B-1 as the real cause. "In all our cases there was the possibility of such local obstruction for every patient had muco purulent expectoration before or immediately after the onset of collapse. We regard this as the essential cause of the phenomena." They accepted Lichtheim's experiments as complete and tried several nerve resections of phrenic and other respiratory nerves in cats. No collapse occurred. Accepting the bronchial plug as the actual cause in their 11 cases, they admit the possibility of other causes. "The eleven cases which we observed agreed closely with Pasteur's description. One feature he laid little stress upon and that we regard as critical. All had mucopurulent expectoration and fever (101 to 103) preceding the collapse. There is no reason why fever should develop in a simple non-septic case with reflex inhibition and 'active' collapse. The chief criteria are fever, the greenish mucopurulent sputum and the displacement of the heart. These together give the distinction from pneumonia or pulmonary embolism, the latter disaster being suggested, in the worst cases, by the acute onset of pain and cyanosis." Previously (1908) MacCallum,[141] taking a leaf from Lichtheim's book, developed a very ingenious technique by which he was able to introduce a pea into a bronchus, producing atelectasis. By a similar technique he produced partial obstruction with short sections of rubber tubing fitting within the bronchus, and produced obstructive emphysema.

To quote or even enumerate the supporters of the mucous plug theory, since Elliott and Dingley, would be burdensome. Pasteur[161] quotes them in the same year with scant approval, and apparently held in this last paper much the same opinion, expressed three years previously[160] that "the early occurrence of viscid expectoration in most cases deserves notice. The sputum at first probably consists of the contents of the collapsing lobe which are pressed back into the large bronchi and coughed up. But if expectoration continues for more than 24 hours it probably indicates the supervention of bronchitis or broncho-pneumonia in the re-expanding lobe or in some other part of the lungs."

Recently, 1928, Coryllos and Birnbaum[41] have thoroughly refined the experimental work of Lichtheim. Working with a collapsible rubber "balloon" they have been able not only to produce bronchial obstruction at will but to relieve it as readily or, in other cases, to experience spontaneous cure. For the balloon they use a piece of closed end rubber tubing, that provided as a cover for the Kollman urethral dilator, and cover it with a limiting bag made from organdie. Fitted to a short section of glass tubing, which carries a one-way valve, the balloon is introduced through a bronchoscope and, *in situ*, filled with potassium bromide solution. It is thus rendered visible for roentgenscopic control. The retaining valve may allow slow leakage and thus the effect of spontaneous cure by coughing the partially emptied obstruction.

Coryllos and Birnbaum conclude after "a careful study of the clinical symptoms, the physical phenomena, the roentgen-ray observations on serial pictures and the pathologic and bacteriologic condition of our animals convinced us that there is but one cause in the production of collapse of the lungs, and that is the more or less temporary, but complete, occlusion of a bronchus by a plug of mucus acting as a foreign body, and intercepting completely the penetration of air into the portion of lung depending on that bronchus. Complete obstruction is an indispensable factor for the production of this syndrome. The other causes in its etiology act only as predisposing factors; the only actual cause being, in our opinion, the complete occlusion of a bronchus."

Lee, Ravdin, Tucker, and Pendergrass,[121] working simultaneously with Coryllos and Birnbaum but independently, have introduced into the bronchus of a dog a mucous mass or plug removed from a man by the bronchoscope. They have also used gum arabic solution of similar consistency and have secured typical collapse by both methods. It was first necessary to inhibit the cough reflex and this was done by intraperitoneal injections of amytal, to avoid any interference with the bronchial surfaces such as might arise from other cough sedatives.

The fairly unanimous acceptance of the mucous plug theory (there is probably now no writer who does not admit that, in at least some instance, it is the terminal cause) is so well presented by Mastics, Spittler and McNamee[140] that I repeat it here.

"1. Physical examination of patients immediately following operation and at various intervals thereafter has shown that atelectasis does not occur until several hours have elapsed. This argues against the sudden production of atelectasis by means of a reflex vagus mechanism and shows that time must elapse during which there is an accumulation of mucus leading to bronchial obstruction.

"2. In a number of instances, we have been able to anticipate the development of atelectasis in patients under anesthesia, who gave evidence of increased secretion in the respiratory tract. We have applied the term 'wet patients' to these cases.

"3. The finding of mucus plugs by Jackson and Lee in two cases at necropsy, and the demonstration of such plugs in vivo. Nearly all observers have noted the presence of mucopurulent expectoration.

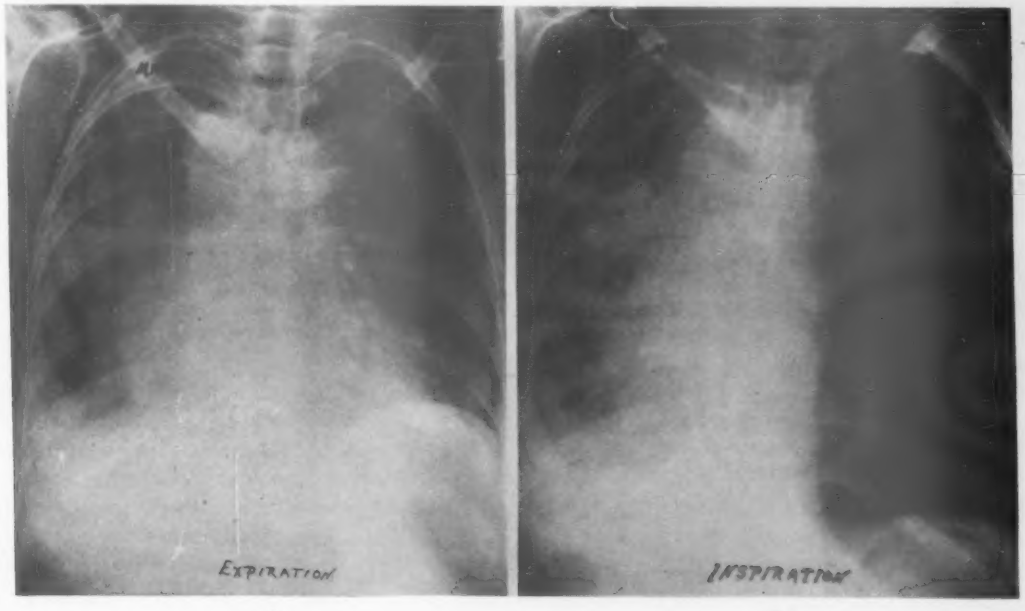
"4. Reinflation of the atelectatic lung following bronchoscopic aspiration of the mucus, as described by Tucker and Manges.

"5. Recurrence of atelectasis following reaccumulation of mucus in the bronchial tree, in amounts sufficient to produce obstruction. The cycle of secretion-obstruction-atelectasis-aspiration-reinflation has been completed seven times in one case, reported by Hearn and Clerf. It would be difficult to explain this phenomenon on the basis of stimulation of the vagus."

Convincing as has been this research work and clinical observation, we may be sure that the conclusions quoted will not be everywhere accepted without question. Sante[177,178] believes with Bradford[21] that autopsied collapse patients in whom no such accumulation was found make acceptance of this theory as the only cause, nearly or quite impossible. Gwyn[70,71] cites "Dr. Wookey's case" the trachea in which became occluded with food during anesthesia, with immediate death, despite efforts at artificial respiration. Bergamini and Shepard[16] report two sudden deaths immediately after operation (one of these also reported by Santee[180]). In these 3 cases early autopsy showed both lungs totally collapsed. It is argued by these

observers that the time element was too short for so complete a collapse to be accomplished by air absorption. The published records of all three cases lack many desired details, but there are two possible explanations. In any case, it seems quite

Golden[67] report similar cases. Junghagen[113] reports a case from the pressure of enlarged "granulomatonic" glands, and one from the pressure of an aneurysm. Wessler and Jaches[215] record cases from obstruction by tuberculous nodes and by bronchial



A B

Fig. 4. M. W., female, aged forty-three; referred for roentgen therapy by Dr. B. M. Allen; metastatic malignancy of both lungs; massive collapse of right lung, presumably from bronchial obstruction. A—Expiration; B—inspiration. Patient at this time showed but little more than ordinary dyspnea, but this rapidly became worse and she was unable to have further studies. Death occurred about six weeks later.

certain that collapse must be well begun before objective symptoms arise and, in addition, it seems very probable that, with suffocation well toward accomplishment, we may expect overactive circulation to absorb the residual pulmonary air with much more than usual rapidity. However, I do not presume to have solved this and other mentioned problems. With all my enthusiasm for the obstruction theory I am not yet willing to demand that it be accepted as the only possible cause.

Acceptance of the mucous plug theory entails the expectation of massive collapse from any occlusion of a bronchus. This is repeatedly attested to by Jackson and his coworkers with bronchial foreign bodies both organic and inorganic. A case of massive collapse from bronchial occlusion by metastatic malignancy of the lung is shown in Figure 4, and Ashbury[5] and fibroma. Manges reports a typical case of massive collapse occurring from mucous plug during one of the paroxysms in a patient with chronic asthma.

I do elect, for now, to consider all causes not above mentioned as predisposing rather than actual. Chief among these is bronchitis. Nasal and pharyngeal catarrh and bronchitis are ever with us. Either latent or active, few people in the so-called temperate zones are wholly free from them. Even of those otherwise well situated hygienically, the great majority will spend a major part of their lives in an atmosphere over-heated and over-dried. The "makings" of acute bronchitis are ever at hand, needing only a temporary inhibition of the protective factors, which have ordinarily prevented their activity. These protective factors are too often interfered with or neglected in the preoperative

hours. (See Prophylaxis, especially quotation from Whipple.)

Morphine and Atropine. Despite the lack of unfavorable opinion and with due respect to a well established practice, I cannot avoid a conclusion that the preanesthetic use of morphine and atropine has a real etiologic relation to the occurrence of massive collapse.

Jackson[99,103] has repeatedly spoken of cough as the "watch dog" of the respiratory system. "There are certain circumstances in which normal agencies are inefficient in ridding the bronchial system of excess secretion. Various drugs, and especially antibechics, hinder the action of the normal agencies; hence should always be avoided. The writer has always opposed their use in all laryngeal and tracheal surgery and in bronchoscopy. Doubtless, many of the post anesthetic pneumonias, in surgery remote from the air passages, have been due to the abolition of the agencies by which secretions are normally removed from the air passages."

"Does it not seem that we increase the risk of post-operative pneumonia by the preliminary or post-operative use of morphia in surgical cases requiring a general anesthesia?"

One surgical colleague agrees with me verbally and continues to use morphine and atropine routinely. Other friends express even less interest in my suspicion. The literature affords the scantiest detail. Scarcely any case histories mention such administration, though its use routinely seems to be all but universal. Lord[129] says: "Probably administration of morphine after operation favors collapse by diminishing the impulse to cough." Hewlett[86] says: "Morphine and other opiates are usually to be avoided during bronchial obstruction and atelectasis. They decrease the tendency to cough, etc." Not even to this extent do I find any written suspicion that the preoperative administration might be a factor. Indeed, the only mention noted, which exceeds two lines, is this from Pasteur:[161] "It is instructive to learn that since the practice of giving an injection of morphine and atropine before anesthesia has become general at the Middlesex Hospital, there has been a striking disappearance of respiratory difficulty (hypersecretion and aspiration) during anesthesia, and a corresponding diminution of postoperative bronchial complications after major operations." Under treatment, Mason[139] thinks morphine should be given to control pain incident to the operation and Gwyn[71] says that "in the majority of cases the use of morphine in large doses is imperative." Nevertheless, and with such cold comfort from others, I shall continue to "think atelectasis" whenever this routine is mentioned.

Anesthetics. That massive collapse of the lung may occur when only a local anesthetic has been used, that many cases have so occurred and that the condition may occur when no anesthetic has been used is adequately proven. It seems to me that this by no means proves that general anesthesia may be absolved from all suspicion as an etiologic factor. One who has over a period of years watched the administration of ether anesthesia cannot fail to remember case after case in which secretion was profuse, the patient giving every sign of respiratory irritation. Mastics, Spittler and McNamee[140], Lee[117,119] and many others have sensed the added risk in these paients. Pasteur[161] absolved the anesthetic but, so to speak, placed it in bad company. "We may, I think, conclude that (1) ether, *per se*, is probably not a direct cause of pulmonary inflammation, and (2) that respiratory troubles during anesthesia, whether due to faulty administration, unsuitable method, surgical manipulation or the quality of the anesthetic itself, favor the aspiration of saliva, etc., into the lungs, and that in this way the anesthetic may become an important contributing cause of lung trouble. I have dealt mainly with ether in speaking of after effects because it has, more than any other anesthetic, been held responsible for them; but as already mentioned, I attach much more importance to disturbance of respiration during anesthesia than to the anesthesia itself." At any rate, it seems that the best interests of patients in general will be served if the anesthetic and the anesthetist are not too generously absolved from all responsibility for this accident. Patients experiencing the so-called "wet anes-

thesia" should be watched with especial care, during the early postoperative period, in order that no premonitory sign of collapse shall be missed.

The Operation. While massive collapse does indubitably follow certain operations with greater recognized frequency, I refuse to see in that any convincing argument for the theory of reflex.

Scott and Joelson[186] have done much to clear the atmosphere by pointing out that in some cases, at least, these operations entail a naturally selected postoperative posture, and that this posture may be one of the causes of, say, contralateral collapse. Further observation seems certain to amplify these findings. It has occurred from so trivial and remote an operation as circumcision,[30] and despite the oft repeated contrary statement, it has occurred after tonsillectomy,[197] and it is by no means proven that collapse is not one of the antecedents of post-tonsillar lung abscess.

There seems to have been no systematic roentgen-ray study of post-tonsillectomy patients, nor any systematic study of the premonitory signs and onset of post-tonsillar lung abscess.

Following the findings of Scott and Joelson we note that the after care and the naturally assumed postures of these tonsil patients all tend to counteract collapse. They roll and toss, sit up to drink and for the commode, and no nursing advice nor admonition will prevent them from rather forcefully clearing the throat. Among the exceptional patients who do not do these things there will be found cases of partial and massive collapse and this will very possibly come to be listed as one or even the chief cause of post-tonsillar lung abscess, soon after the tonsil surgeons begin to "think atelectasis."

TYPE (SYMPTOMATIC)

The condition may be considered as: (1) massive, involving a lobe or lung, and (2) partial, lobular or "patchy" when smaller areas are involved.

The latter type has an especial importance (Lee[117]), in that it almost certainly affords frequently a lodging for minute emboli which, as Cutler[46] has shown, are a constant result of all operative procedures in which blood vessels are opened. Mastics, Spittler and McNamee[140] suggest the following classification:

Fulminant; with sudden onset and a stormy course ending by crisis, lysis or with complications.

Moderate; with fairly well marked differential signs but of distinctly lessened severity.

Latent; with little or no subjective symptoms but demonstrable clinically by physical examination and by roentgen-ray appearance.

Evanescent; without subjective symptoms and with physical signs suggestive only or more commonly undiscovered, but demonstrable by roentgen ray.

Of little practical importance, this type goes, and perhaps comes again, with change of position, deep breathing, etc. It doubtless bears a very close relation to the physiologic atelectasis of Sewall,[190] Webb, Forster and Houck,[211] and others.

SYMPTOMATOLOGY

In by far the greater number of cases a rise in temperature is the first symptom of impending collapse. This rarely exceeds 102°F., and is probably at that time due more to disturbance of the heat regulating function of the lung than to any other factor. Any rise of temperature, after the postoperative reaction is over, should lead the physician to "think atelectasis." If the chest be examined at the time of this premonitory rise in temperature it is probable that bronchovesicular signs of importance will be found. In the fulminant type this rise of temperature is rapidly followed by what Lee calls the "catastrophe." To one witnessing, for the first time, one of these attacks a lesser word seems most inadequate. The face is suffused and the expression anxious. Dyspnea is urgent and there is marked diaphoresis. The patient complains of a sense of tightness commonly amounting to real intrathoracic pain. There is ordinarily a gradual remission of the urgent symptoms and, to some extent at least, this is coincident with a returning confidence on the part of the patient. Commonly a sort of equilibrium is established within a few hours and from that time the course, even if frank and unmasked, is less urgent. The dyspnea

is better borne, the erythema and diaphoresis is less marked, and the patient assumes a characteristic position with the head toward the affected side. Pain continues but is deep-seated in character, quite unlike that of pleurisy.

It is this urgent type, with every sensation a demand for relief, that prompts bronchoscopy and in which bronchoscopy gains its most spectacular result.

The onset may vary from this to a beginning so mild that only careful routine will detect it. Increased temperature, pulse and respiration rate are rarely absent. Cough in some degree is commonly present but in the early stages is suppressed in character. Expectoration is scanty, "gum drop" appearing, becoming mucopurulent and more profuse beginning with the abatement of symptoms. The crisis type of recovery is nearly always accompanied by an increase in expectoration, so much so that we have used the term, "recovery by expectoration."

The date of incidence is frequently within twenty-four hours and commonly within seventy-two hours, though so long an interval as one week or ten days is occasionally reported. The site of election has been reported as the right lower lobe, though Crymble[43] found the larger number of his series in the lower left lobe. Crymble's cases were war wounded, several of them with contralateral collapse. Contralateral collapse was common during the war and is not uncommon among the postoperative cases. This feature has appealed to some as a support of the theory of reflex etiology, but I believe that Scott and Joelson[186] offer a more credible theory in asserting that the position which the patient assumes because of the discomfort following herniorrhaphy, nephrectomy or other operation, is the largest factor in determining the site of collapse and, therefore, in producing this contralateral phenomenon.

FREQUENCY OF OCCURRENCE

This is admittedly a difficult question. We most certainly cannot believe that massive collapse occurs with confessed frequency in certain hospitals while in others, with practically the same percentage of postoperative pulmonary complications, it does not occur at all. We persist in believing that the latter hospitals have not yet begun to "think atelectasis." Cutler[46] estimates the incidence of postoperative lung complications as "one in every 30 to 50," while Mastics, Spittler and McNamee[140] believe that approximately 70 per cent of all such complications are atelectasis. Lord[129] found 14 in 558 cases of pneumonia. Gwyn[71,72] believes that "acute collapse as a complication of pneumonia has been under our eyes from all times." The teaching of all this is that massive collapse occurs with sufficient frequency to make it distinctly important to "think atelectasis" whenever there is any postoperative lung complication, to say nothing of its occurrence in lung disease generally.

PHYSICAL SIGNS

Perhaps no other known condition affords such striking examples of the sometimes wide difference between physical examination and physical diagnosis; between the careful elicitation of physical signs and the formation of an accurate opinion from the interpretation of those signs. Sewall[190] says that: "It is highly probable that the condition of atelectasis as such is seldom recognized. Only when the examiner definitely *thinks* atelectasis will its signs usually be correctly apprehended and will then be common enough. In the psychology of physical diagnosis nothing is more certain than that our apprehension of a patient's condition depends not upon what we know of physical signs, but upon what we consciously apply to the subject under investigation. If the examiner but thinks atelectasis, bears in mind the conditions of its occurrence and applies the simple tests for its recognition, physical diagnosis of the chest will gain much in certainty."

One must begin with a realization that only in typical cases will there be typical signs. All physical signs will be modified by the position and size of the collapsed area. In the severe cases the distress of the patient and his actual, though perhaps unexpressed, resistance to physical disturbance must be taken into account. For this reason, as well as for merit inherent,

inspection is highly important. The patient does not lie straightened out in his bed but with the thorax contracted and fixed on the affected side. The corresponding shoulder will be depressed toward the hip while the opposite shoulder will be correspondingly elevated. If the patient is spare, depression of the intercostal spaces can be seen; also their decreased width as compared with the uninvolved side. The decreased excursion of the diaphragm on the affected side, more commonly its fixation, should be noted. Cyanosis usually described as brick red, and the labored type of inspiration are striking. Present but perhaps less easy of detection is the lessened range of chest movement. We note with Scrimger[188, 189] that we have not seen immobility nor was the position assumed even that of extreme expiration. *Palpation* with one hand on each side of the chest will occasionally detect lessened range of chest movement when the decrease is not enough to visualize. Vocal fremitus will be subject to variations in accord with the auscultation and percussion sounds. If obstruction is complete the vibrations are absent or present to a very slight extent. In "drowned lung there may be some transmission of the vibrations but this is usually slight" (McCrae[145]). *Auscultation* and *percussion* signs will be variable in conformity to the many variations of location, area and the condition and position of adjacent lung parts. Auscultation will vary from entire absence of sounds over a wholly collapsed lung to many, and sometimes paradoxical, râles when the adjacent parts of one lobe are in partial collapse or when a collapsed lobe is overlaid by an uninvolved lobe margin. This is particularly noted when involvement is confined to the lower right lobe. Similarly percussion will disclose large areas of flatness or small areas of unimpressive dullness. Sewall[190] warns that; "The physical examiner is wont to over-estimate the depth beneath the chest wall to which percussion and asucultation extend their informing signs. It is not unknown for the *x*-ray plate to disclose a relatively huge mass occupying the center of the lung, the existence of which has been missed by expert examiners. According to Weil,[213] where the chest wall is moderately thick, no percussion vibration can penetrate the lung for more than 4 cm. (1⅗ in.) in a vertical direction." By far the most important characteristic sign is the displacement of the heart toward the involved lung. Pasteur,[161] Jackson and Lee,[109] and many others say that the condition cannot be diagnosed with certainty unless this sign is present. Ordinarily lateral, it may in case of left-sided collapse be displaced upward well toward the axilla, but it is in the presence of massive collapse in the right lung that the greatest heart displacement occurs. After fever displacement may be almost the only sign. A patient seen in the Pennsylvania Hospital in June, 1928, not otherwise included here, showed displacement unmistakably with very few other signs. He was studied because of a slight elevation of temperature with very slight dyspnea. The only additional roentgen sign was a faint shadow at the left cardiophrenic angle. By itself this shadow could not have been positively distinguished from that given by a small pleural effusion; yet the heart displacement was unmistakable and particularly so on the following day when the case was fairly frank by other signs.

To the physician wholly unacquainted with massive collapse the greatest difficulty arises in the effort to understand the mechanism of heart displacement. To rid the mind of a lifelong conception of lung collapse as a phase of pneumothorax appears often to give rise to real difficulty (See footnote page 105).

It is true that in the early stages the actual condition of the lung involved with massive collapse may not differ greatly from that of another lung, collapsed because of pneumothorax, but this applies to the lung only. The effect upon all other organs of the thorax is vastly different. In both conditions the lung is collapsed, though the massive collapse here considered only rarely involves an entire lung; but in massive collapse the atelectasis has proceeded from causes within the lung and to some extent the negative pressure is countered by sequences within the lung, as hypersecretion and engorgement. In pneumothorax, on the other hand, the

lung participation is wholly passive, the air being forced out by increased pressure upon the surface of the lung.

In pneumothorax, the space normally occupied by the now collapsed lung, is filled by foreign air ballooning the pleural cavity. In massive collapse this compensation is rendered by at least five factors mentioned here in what is perhaps the reverse order of their importance, viz., hypersecretion, circulatory engorgement, depression of the thoracic wall, elevation of the diaphragm and, most important of all, emphysema of the unaffected lung. Until these essential differences are clear, and until the potential mobility of the mediastinum (Sewall[191]) is adequately valued, one cannot by any possibility be in the way of understanding massive collapse of the lung.

The displacement of the heart is, therefore, due directly to the negative pressure of the affected side where the collapsed lung, or lung part, is occupying less than its normal area, leaving a space to be filled by whatever may be easier to get, and just as directly to the enlarged, from emphysema, unaffected lung. There being no inspiratory expansion of the collapsed portion, all movement within the thorax must coordinate with the emphysematous opposite lung. The heart displacement, being thus responsive to the emphysematous lung, will be greater when the emphysema is greatest, i.e., on inspiration, and less when the emphysema is least, i.e., on expiration; hence the lateral excursion mentioned elsewhere. With the possible exception of Samuel West,[217] no one preceding Pasteur had noticed this sign. Stokes[200] noted mediastinal displacement but did not offer cardiac displacement as a diagnostic sign. Hewitt[84] and Weil[213] (1871 and 1880), cannot be excused for omitting to mention this sign, if they knew it. Whipple[218] and Cutler and Hunt[44] must have missed it in their excellent presentations of postoperative lung conditions for they do not mention it. Gwyn,[71,72] discussing 250 cases of postoperative pulmonary involvement recently analyzed, says: "There was but one note that said that the heart was in position. Yet, many acutely developing cases of unilateral consolidation were being considered." Griffith,[70] a pediatrist of unusual experience and acumen, says that "displacement of the heart is not always discoverable on percussion due to possible emphysematous distention of adjacent edges of the lung.The ultimate diagnosis is believed to rest upon the radiographic appearance." It is somewhat unfair to have mentioned these and to have omitted literally dozens of others. As a matter of fact, here is a phase of physical diagnosis pertinently comparable to violin playing, impossible, difficult or pleasurably easy in direct accord with amount of teaching and experience, to say nothing of talent, which one may bring to bear upon it.

Roentgen-ray diagnosis of this condition will be considered, as I believe it always should be, as just another method of physical diagnosis. As in so many other fields, so here, roentgen diagnosis achieves its greatest reward when co-operation with other methods is most hearty. The roentgenologist is wholly dependent for his opportunity upon the attending physician or surgeon and, until these begin to "think atelectasis," he will see only the cases which are, more or less by accident, referred because of a supposed diagnosis.

It is doubtless this fact which largely accounts for the paucity of writing on this topic by roentgenologists.

The following table (Table 1) of comparison of the affected and the unaffected lungs is repeated here for convenience.*

The condition is best recognized by the roentgenoscope and to this fact may be ascribed the tardy appearance of roentgen methods in the study of collapse. Surgeons

TABLE 1

ROENTGEN SIGNS OF MASSIVE COLLAPSE (ATELECTASIS) OF THE LUNG

Affected Lung		Opposite Lung
Increased	1. Density	Decreased
Displaced toward	2. Heart and trachea	Displaced from
Elevated	3. Diaphragm	Depressed
Decreased	4. Intercostal spaces	Increased
Decreased	5. Phrenic and costal excursion	Increased
Curved from	6. Vertebrae	Curved toward

Signs from 2 to 5, inclusive, are accentuated by inspiration and lessened by expiration; in other words, a pendulum-like excursion of the heart with respiration.

Entire absence of this feature should cause a search for complications or a new diagnosis.

* Am. J. ROENTGENOL & RAD. THERAPY, April, 1928, xix, 360. Unfortunately No. 6 was reversed and should be as here printed.

were, and still are, loath to permit recently operated patients to be taken to the roentgenoscope. At the Pennsylvania Hospital we had developed a technique for this purpose and, from a date preceding our first recognized case of collapse by some two or three years, it has been used freely by the entire staff.

An iron carriage with wheels for each corner leg, having no central wheel or other structure, has been constructed to carry a separate wood-frame, canvas-top stretcher. This stretcher having been covered with the needed blankets, the patient is lifted vertically from the bed by four attendants who have a firm grasp on the sides of the under sheet. While the patient is thus held above the bed the blanket-covered stretcher is passed under him and he is lowered onto it. The stretcher is then lifted to the carriage and later from that onto the horizontal roentgenoscope or in front of the vertical one, as the desire may be. No manipulation of the patient is needful unless it be rotation (still reclining) at the vertical roentgenoscope. Back at his bedside the loading process is reversed. Surely no adequate physical examination can be made with less distu bance.

As in other physical methods, the one outstanding sign is the displaced heart. In many cases this may be easily missed if one does not definitely look for it. It is best detected by comparing deep expiration with deep inspiration and the two with some fixed point on the screen, as the margin of the shutter shadow. In no other condition with which I am familiar is there any appreciable *lateral* movement, though an upward movement of the apex may be considered to normally accompany inspiration. Manges[135] calls attention to "times when the heart seems to swing as if it were a pendulum swinging from the upper mediastinum, and at other times it seems that the entire mediastinum, both upper and lower, moves laterally to almost the same degree."

Films, if made, should be in both phases of respiration—full inspiration and full expiration. A single film or even a stereo pair, made with the usual admonition to "take in your breath and hold it" may have scant value. If the lesion is less than lobar a lateral film, made, of course, with the affected side toward the film may be useful for localization.

The density of the affected lung has caused some discussion. Sante[177] writes: "When one considers the small size to which a normal lung may collapse in the presence of pneumothorax and its relative radiolucency when fully collapsed, it is surprising that in this condition a fully collapsed lung could occupy so much space and acquire the marked density that it does. The radiographic density of the lung in massive collapse is fully as great as that seen in lobar pneumonia." And again: "Density in contrast to the almost transparent structure of lung collapsed to even smaller size from pneumothorax, is difficult to understand and has never been successfully explained;" and Scrimger:[188] "The degree of density to the *x*-ray was surprising in the absence of consolidation or fluid, and may be taken to some extent as evidence for the airlessness and compactness of the collapsed lung. The sudden relief of symptoms experienced by three of the seven, with the rapid aeration and expansion of the lung as shown by the return of resonance over large areas, the return of the heart to its normal position within a period of at most 6 hours in all three, and 2 hours in one, preclude the possibility that the density is due to consolidation or the collection of fluid."

For a long time I held firmly the opinion that this density was, nevertheless, fluid; "drowned lung." This explanation of the density has occurred to others, particularly Churchill,[34] Leopold[125] and Mastics, Spittler and McNamee.[140] Later I realized that drowned lung, so frequent in foreign body atelectasis, is in reality rarely seen, in any large degree, in any other massive collapse.

In a recent conversation, Dr. Sante pointed out what seems to be the real source of this density, i.e., engorgement of the vascular system. This is, of course, logical. We have an active potential vacuum sufficient to depress intercostal muscles, elevate the diaphragm and displace the mediastinum. An immediate source of partial relief is the vascular system. The blood and lymph channels become en-

gorged as readily as do the subcutaneous channels when a patient is "dry cupped." When the potential vacuum finds other relief, i.e., normal aeration, the congestion disappears quickly and the density with it. I have not found this explanation in print.

Just how collapse can occur with complete absence of secretion or engorgement is more difficult of explanation; yet in at least one case, Chizzola,[32] this seems to have happened.

I remember many of my own reports to have read something like this: Such or such a shadow was distinctly seen in the fluoroscope but "nothing of it is seen in the stereoscopic films." This was probably the physiologic atelectasis described by Sewall[190] and by Webb.[211,212] The patient had been roentgenoscoped lying on the above described stretcher in the same position which he had occupied in his bed, but when the films were made he was momentarily sitting erect and the exposures were made at inspiration. (This refers, of course, to patients with conditions other than massive collapse.)

Finally, once a group begins to "think atelectasis" an unexpected number of cases are seen in which partial collapse seems rather certain but remains unproven even by roentgen ray. There may be an indefinite shadow but no heart displacement to be positive about. It seems to me that such findings, with prompt recovery, are probably minor degrees of collapse. Demanding increased vigilance in observation these cases seem otherwise rather unimportant.

DIFFERENTIAL DIAGNOSIS

The most common error of diagnosis in the presence of massive collapse is pneumonia. The two conditions have so much in common that Coryllos and Birnbaum[41] propose the theory that they are mechanically the same, it being needed only that the infection of the preceding bronchitis shall, in the case of pneumonia, be the pneumococcus. We have seen under "symptomatology" that collapse may at the least appear not rarely as a complication. Generally, however, the heart displacement, the absence of bloody or rusty sputum, with the lower temperature and pulse rate will aid the decision. Jackson and Lee[109] believe that the reason massive collapse of the lungs is so often overlooked is that its mere presence is at once regarded as conclusive evidence of the existence of pneumonic consolidation. Due regard to the other signs, particularly cardiac displacement, should make the diagnosis clear. In other words, "think atelectasis."

Bronchopneumonia has, from the very beginning, raised great differential difficulties. Gairdner wrote: "The judicious physician has doubtless often been saved from error in the application of his remedies in this, as in many other cases where an unsound pathology has prevailed, by adhering to the great principles of our art, and the teaching of personal experience, rather than to received formulas and rules on matters of detail. But it is difficult to escape from the conviction that the influence of a name, or the vanity of an exact diagnosis, may have misled many into such errors; and that in cases of supposed bronchopneumonia or typhoid pneumonia, the practice may frequently have embraced blood-letting, heroic antimonials, or calomel and opium, when emetics and expectorants, with suitable stimulation, would have been better adapted to the circumstances of the case. This will be still more evident on considering the mechanism of these affections." And from this ten years further back to Williams: "Nothing can show more forcibly the influence of a name than the fact that this condition of the lungs should have been described by all writers as lobular pneumonia, and that its symptoms should have been attributed to inflammation, while yet it was evident from the concurrent testimony of everyone that neither in its progress nor in its results was it similar to inflammation of the lungs in the adult, much less identical with it. Having, however, once been called pneumonia, every person continued to call it so, though often with a full recognition of its peculiarities. Even the close resemblance which the lung presented to foetal lung, or to those undilated portions which are characteristic of atelectasis was noticed and discussed by myself and by many far better observers, apparently without a suspicion that both states were identical."

For more recent evidence of the need for "thinking atelectasis" we may cite the report of Hampton and Wharton,[76] an analysis of 170 cases with 80 diagnoses of pneumonia and bronchopneumonia and 34 of infarct with only 5 deaths, in none called pulmonary infarct, the only word about collapse being, "it has not been observed in this clinic." Yet Cases 1, 3, 4 and 5 show roentgenograms nearly enough typical to warrant a challenge.*

We may, with scant fear of error, take

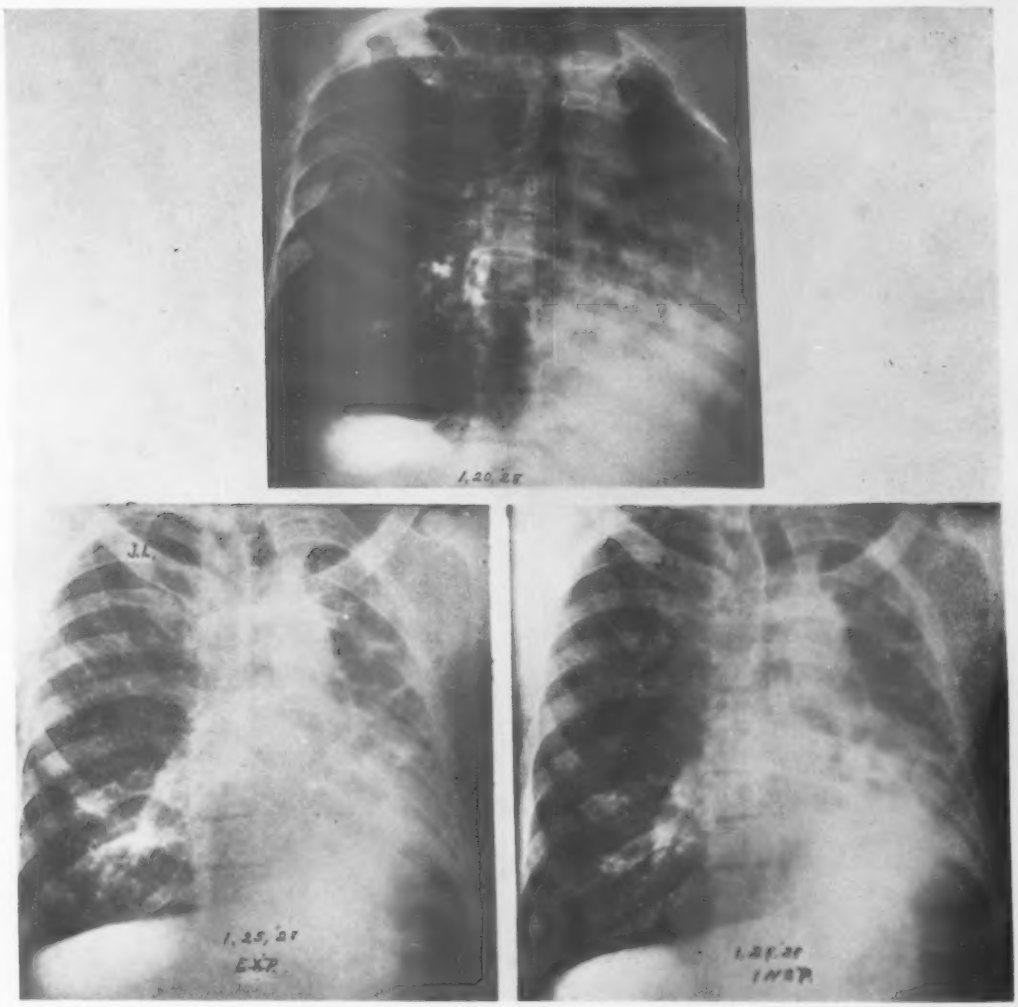


Fig. 5. J. L., male, colored, aged forty-three; came to receiving ward with acute attack, symptomatically, of massive collapse. Roentgenoscopically, marked left displacement, no excursion. Film at this time and films made at inspiration and expiration five days later are almost identical. Case proved to be chronic fibroid tuberculosis. Patient probably had acute bronchial obstruction at the time of admission.

of which was the final diagnosis pneumonia. In 10 per cent a first infarction was followed by a second. It is by no means inferred that all of these should have been recognized as collapse but it is equally as certain that some of them should. Or again in the same year the paper of Cutler and Hunt[44] in which 42 of 63 cases were it for granted that every observer of postoperative lung complications will, once having recognized massive collapse, realize that many puzzling cases are easily explained in the light of the newer experience, while many diagnoses, eminently

* Both Dr. Wharton and Dr. Cutler have, in recent letters, cordially accepted this criticism, each having arrived at a similar conclusion from later observation.

satisfactory a few years ago, are now readily recognized as errors.

Pneumothorax may be suggested by hyper-resonance of the opposite lung and subphrenic abscess by the high position and immobility of the diaphragm.

was made. "The patient cried much and breathed deeply during the operation; and immediately thereafter percussion of the right chest disclosed a dull tympany so that, for a while, it was feared that I had caused a traumatic pneumothorax. The

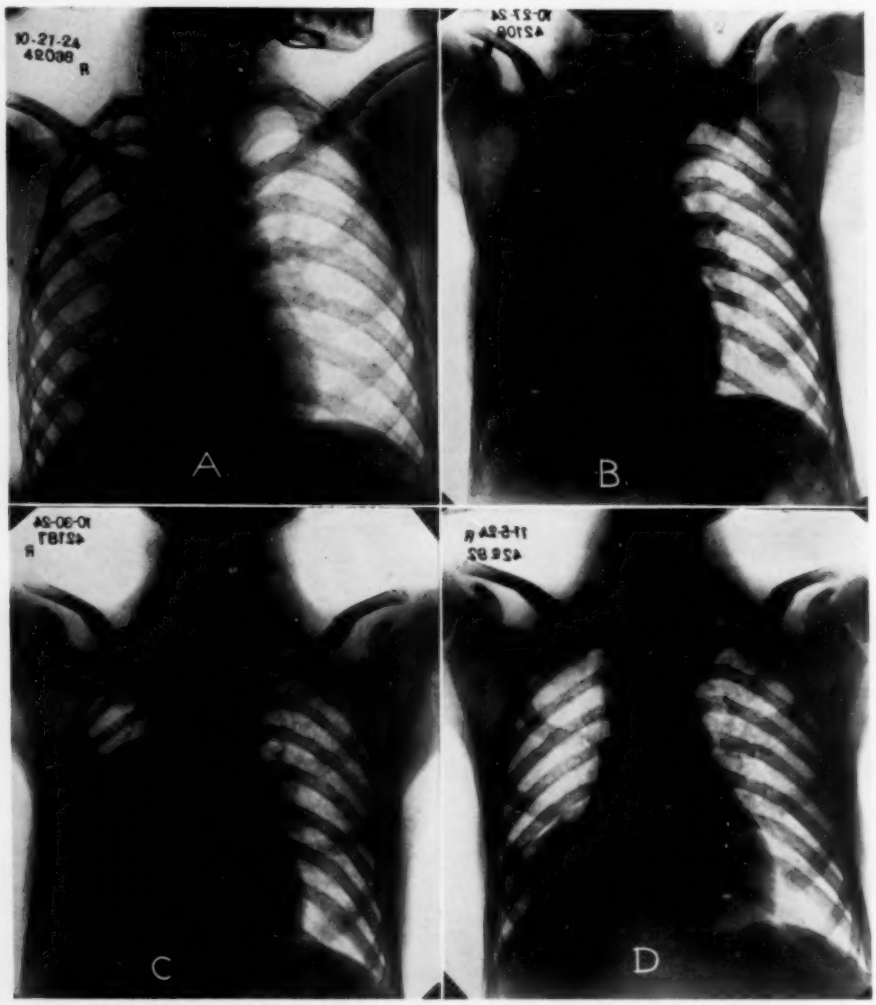


FIG. 6. Typical massive collapse without special treatment. I. P., undernourished Jewish boy, aged fourteen; emergency appendectomy; right collapse, fifth day postoperative. A—First day, collapse partial. B—Seventh day, collapse complete. C—Ninth day, upper lobes clearing. D—Seventeenth day, collapse confined to lower lobe. Recovery was complete on the twenty-sixth day.

Embolism is doubtless far more rare as a gross demonstrable lesion of the lung than has been supposed. It does not cause mediastinal displacement.

Pleural effusion has been repeatedly suspected when the real lesion was collapse. Sewall[190] records a case in which a "dry tap" of a supposed right pleural effusion boy got well with diminishing signs of effusion." (Presumably a recovery very similar to those described by Sante.)

A previous lesion may be the cause of error (see Fig. 5). Finally, we may well agree with Churchill[34] that postoperative massive collapse is "just one special phase of atelectasis as a general subject."

CASE REPORTS

There is here no attempt at case reporting other than the captions which accompany the illustrations. All these cases (Fig. 4 excepted) have been, or will be, elsewhere reported and should not be considered in any future analysis by others.* The following tables give findings from eleven cases observed prior to June, 1928 (Tables II, III and IV).

DURATION

The duration is variously stated as from a few hours to approximately four weeks. One of these illustrated (Fig. 6) continued for nearly or quite three weeks. Sante and others have noted impairment or loss of the diaphragm excursion, the diaphragm in that case seems to remain persistently high for considerable periods after other roentgen-ray signs have disappeared. Though always in mind, this has been seen in none of our cases. Duration is so variable that there will need to be much accumulated experience before the real merits of any therapeutic program can be decided. I have seen a bronchoscoped case recur within twenty-four hours and be relieved permanently by expectoration within six hours after the recurrence.

TABLE II

POSTOPERATIVE MASSIVE COLLAPSE OF THE LUNG

Bronchoscopic Data (5 Cases)

Case	Date	Onset, Postoperative	Bronchoscoped; Time after Onset	Material Aspirated	Immediate Effect	Normal Position Heart and Diaphragm Lung Clearing
E. S.	9–10–27	36 hrs.	6 days	4 c.c.	Slight	2 days
D. D.	10–22–27	36 hrs.	48 hrs.	6 c.c.	Slight	8 days
C. M.	1– 9–28	36 hrs.	48 hrs.	9 c.c.	Marked improvement	2 days
E. C.	2–15–28	35 hrs.	5 hrs.	3 c.c.	Marked improvement	2 days
J. G.	5–18–28	38 hrs.	10 hrs.	5 c.c.	Marked improvement	3 days

The first two of these cases had cultures made of aspirated material. (1) showed *Streptococcus hemolyticus;* (2) pneumococcus, Type IV, and *Micrococcus catarrhalis.*

All cases discharged cured at normal time for type of operation,

TABLE III

SYMPTOMS

Pain in chest..... { yes—6 cases; no —5 cases }

Dyspnea.....All

Cyanosis..... { yes—6 cases; no —1 case; not stated—4 cases }

Cough.....All (suppressed in most because of pain).

Temperature, Pulse, Respiration } sharp increase to { 101.4–104.4; 100 –136; 30 –48 }

Previous *Lung* Condition

7 cases negative.
1 case history occasional cough; physical examination, x-ray and laboratory negative.
1 case bronchial asthma.
1 case silicosis (hilum type).
1 case apical tuberculosis (healed?).

TABLE IV

POSTOPERATIVE MASSIVE COLLAPSE OF THE LUNG

Age	Yearly Incidence	Operative Incidence	Time of Onset
No. Cases Yrs.			
1 15	1924—1	Herniorrhaphy—7	30 to 96 hrs.
2 16	1925—0	Appendectomy—3	(postopera-
4 17	1926—1	Nephrectomy —1	tive)
1 18	1927—3	Anesthetic	
1 21	1928	1 local (hernia)	
1 34(6 mos.)	—7	(age 47—silicosis)	
		1 Nitrous oxide	
		9 Nitrous oxide and ether	

Collapse—same side	Recovery Cases Bronchoscoped
Appendectomy —2	3 cases—2 days
L. Herniorrhaphy—1	1 case —3 days
R. Herniorrhaphy—2	1 case —8 days

Collapse—opposite side	Cases not Bronchoscoped
Appendectomy—1	2 cases—10 days
Nephrectomy —1	1 case —11 days
Herniorrhaphy—5	2 cases—12 days
{ 2 lt. 3 rt. }	1 case —18 days

All cases had preoperative dose of morphine and atropine.

PATHOLOGY

If we must confine our studies to postoperative massive collapse the result will be meager indeed. First, because death is rare and with improved diagnosis and treatment it will be even more so. Second, because if death does occur it is after changes which have so altered the primary condition of collapse as to render the findings of scant informative value.

Legendre and Bailly[123] worked exclusively with infants, from the newborn and stillborn to the age of five years. But their findings of gross pathology have been but little, if any, amplified during

* These cases, with the exception of Figure 1 (from Dr. Jackson's Bronchoscopic Clinic, Jefferson Hospital), are all from various services of the Pennsylvania Hospital: Drs. Gibbon, Billings, Mitchell, Lee and McCrae.

the eighty-four years that have since passed. For brevity their findings in atelectasis, as compared with "hepatization" have been here tabulated (Table v).

TABLE V

LEGENDRE AND BAILLY (ADAPTED) (1844)

Atelectasis	Hepatization
Red, as dregs or lees of wine	Red, as mahogany; fawn color
Lobular divisions and interstices apparent. Arterioles occasionally seen.	Surface uniform without anatomical characteristics.
Margin of lesion lobular	Margin of lesion irregular
No pleuritis	Pleuritis in some degree
Non-aerated but soft, pliable as muscle (now called beefy)	Non-aerated but hard to touch like liver
Inflation causes expansion to normal appearance	Inflation causes "cracking"

It was this one last mentioned point, the inflation to normal appearance, which they stressed to differentiate this condition from pneumonia or pneumonitis of any and all forms. They called it "etat fetal" or the fetal state, and recognized the second variety, called the *congested fetal state*.

"Along the length of the posterior border of the lung one can observe a great number of lobules, deprived of air, disseminated in the midst of healthy tissue or grouped in a manner either isolated or joined together. Whether isolated or joined together, these lobules are perfectly circumscribed by cellular intersections. They are red but a little less violet in color than the first form. They are less depressed and more friable; and when they are crushed they allow a considerable quantity of sanguinous fluid without air bubbles to ooze out. Without doubt, the accumulation of mucus in the bronchi opposing the free entry of air into the cells and the weakness of the patient render inspiration less complete and less profound and thus contribute to the development of this second variety. We have seen in cases of edema of the lung following scarlet fever a serous infiltration, extravesicular, which gives the appearance of the hepatization of pneumonia; but when the lung is insufflated these portions again become aerated as in the normal state."

This second variety is, in other words, not greatly different from that which we are calling "drowned lung." The facts of the matter are undoubtedly that lung collapse is lung collapse, whatever its antecedents may be, and that if we wish to study its minute structures, uncomplicated, we may do so whenever a lung specimen from pneumothorax or excessive pleural effusion is available. Otherwise, the material will come at unexpected occasions and at increasingly rare intervals. Corper, Simon and Rensch,[40] and others have shown that the circulation of collapsed lung areas is impaired but little. Their research is borne out by Andrus,[3] who offers the following conclusion:

"Collapse of the left lung, in dogs, through ligation of the left primary bronchus produces the following immediate results:

"1. The collapsed lung still transmits about 30 per cent of the calculated total cardiac output.

"2. There is a decrease of about 42 per cent in the total lung volume.

"3. These two factors cause a reduction of 30 per cent in the carbon dioxide of the expired air, an increase of the carbon dioxide content of the arterial and venous blood, and a decrease of 5.5 per cent by volume (30 per cent) in *arterial* oxygen content.

"To offset these deleterious effects, simultaneous changes of a compensatory nature occur as follows:

"1. The respiratory minute volume and the rate of respiration are increased about 20 per cent.

"2. The pulse rate is increased by 20 per cent, with a corresponding increase in the total cardiac output.

"3. The circulation through the aerated lung is increased by 60 per cent in volume per minute.

"4. Within 24 hours there is marked increase in the red cell count and the oxygen carrying capacity of the blood."

By introducing a wool tampon through a tracheal opening into the right bronchus of a dog, Hoover and Pearce were able to demonstrate that, when the bronchus was plugged, the arterial blood (femoral artery) was much darker. Removal and reinsertion had prompt effect on arterial blood color.

I have found only scant reference to the subject of massive collapse in textbooks of pathology. The following from MacCallum[142] is fairly typical. Speaking of it as a curious condition, with the heart displaced toward the involved lung, as determined by auscultation and roentgen

examination, MacCallum continues: "It appears that in some cases observed at autopsy a dense plug of mucus has been found in the bronchus, and this, of course, would readily explain the collapse."

Pathological Types. Considered pathologically, three, possibly four, groups or types may be differentiated as follows:

(*a*) The paralytic type, or Pasteur Type I is, I believe, a terminal lung condition, ordinarily an event of death, and doubtless occurring just as Pasteur outlined in his 1890 and 1908 theses. It appears that either paralysis or fixation of the diaphragm may be a major etiologic factor. In Pasteur's second (1908) paper it seems that in all the adult cases cited phrenic fixation rather than paralysis was the condition. Similarly the cases cited by Lee and Paul[118] may be classed as due to fixation. The accompanying sketch by Dr. Paul (Fig. 16) depicts an autopsy finding as yet unexplained. The sketch is typical of several, all bilateral rather than as indicated in the figure. Gravity can have no bearing because these patients died recumbent. So far as the cases of Lee and Paul go, the condition appears to accompany death in cases where phrenic fixation and respiratory weakness are both present. That these cases have anything in common with acute massive collapse, as depicted in this thesis, I do not believe. It is, however, the type of Legendre and Bailly and other writers before Gairdner. Jenner[111] found several cases of this type in autopsies after typhus and typhoid fever. He made no effort to explain their causation.

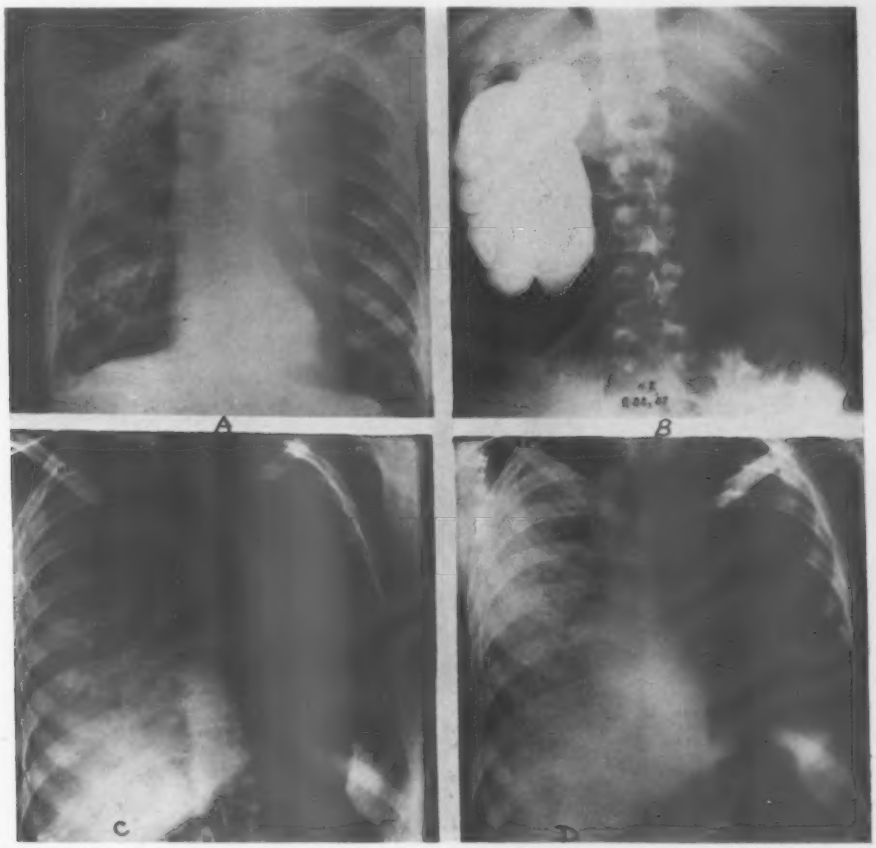


Fig. 7. H. Z., male, aged seventeen; left nephrectomy; right collapse. A—Film one year before operation, showing chronic fibroid tuberculosis. B—*Left* hydronephrosis from congenital anomaly of renal blood vessels. C—First day of collapse forty-eight hours after nephrectomy. D—Second day of collapse, complete.

(*b*) Acute massive lobar or lobular

collapse—the topic of this thesis—the Type II of Pasteur* we have defined elsewhere. This type includes all collapse from bronchial obstruction, without regard to the obstructing agent or force. Ashbury[5] and Golden[67] report cases from malignancy of collapse as occurring in pneumothorax, pleural effusion or from any pressure exerted from outside the lung. Cases of localized collapse have been reported from pressure of dilated heart, thoracic aneurysm and neoplasm.

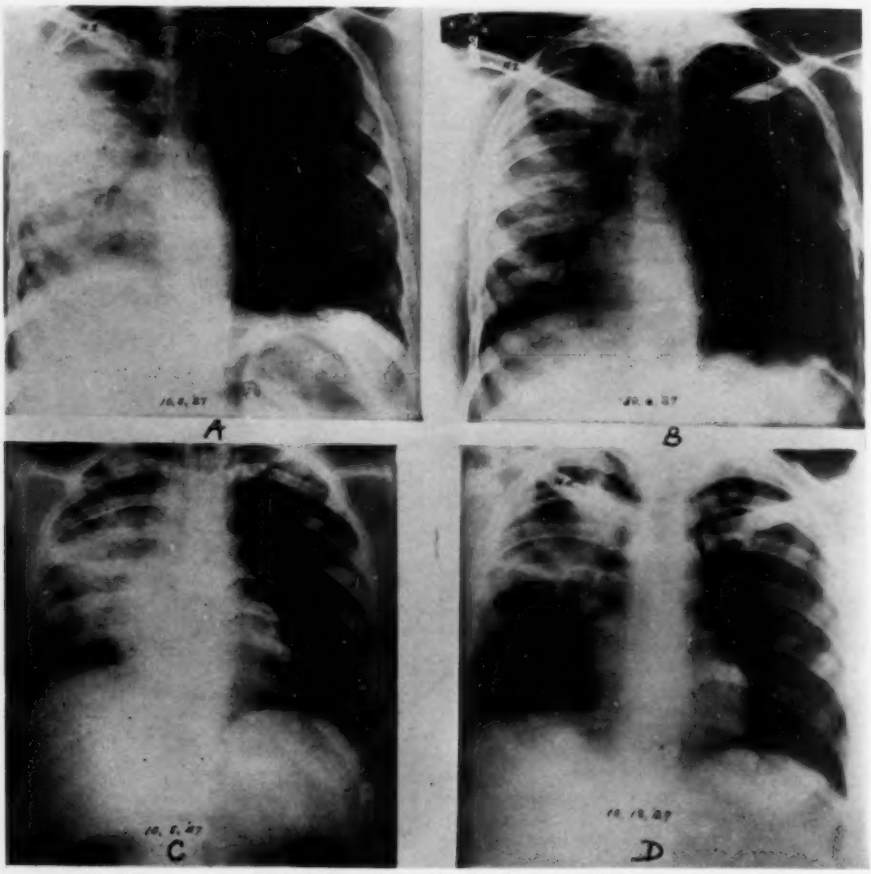


FIG. 8. Same case as in Figure 7. A—Third day slight clearing in upper lobe. B—Fourth day, marked clearing in upper and lower lobes, only slight change in heart position. C—Sixth day, despite greater relative density at hilum, heart is resuming position; remaining collapse, presumably lobular, overlying hilum. Diaphragm still high. D—Eleventh day, nearly or quite to preoperative condition (tuberculosis has remained quiescent since operation).

and I have had one such case (Fig. 4). Junghagen[113] reports one from the pressure upon a bronchus by enlarged glands and one from similar pressure by aneurysm. Speiss[199] reports a case from bronchial polyp. Many other forms of bronchial obstruction might be cited including, of course, all obstructive foreign bodies.

(*c*) In this group are included all cases

(*d*) Three, at least, cases have been reported in which practically instant death has been caused by, or accompanied by, complete collapse of both lungs. One by Bergamini and Shephard[16] and another by Gwyn,[72] "Dr. Wookey's case," appear to have started with choking from vomited food during anesthesia. These histories leave something, at least, to conjecture, but the cases have been cited as too rapid for collapse by absorption of retained air and therefore a point against that hypothe-

* These phrases "Type I and Type II of Pasteur," are borrowed, not from Pasteur but from Powell and Hartley[167a]; also Pasteur did not include lobar and lobular "patchy" collapse in the same classification. I believe their etiology is essentially the same, the difference being entirely one of area.

sis. It seems probable that in such sudden catastrophe the time factor may have been longer than it seemed; that a considerable deflation may have occurred by valve action during the choking and that, during death by choking, there may be a more rapid absorption of air than under other circumstances. It is not at all certain that these cases demand a separate classification.

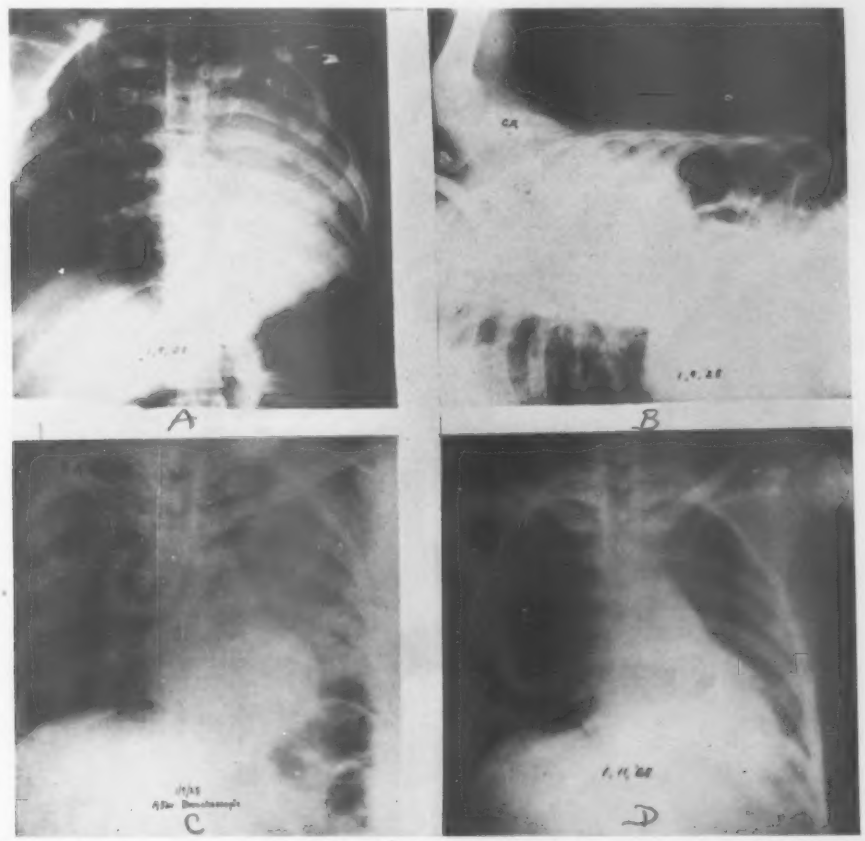


Fig. 9. C. M., male, aged seventeen years. Right herniorrhaphy, left lung collapse thirty-six hours after operation. A—Two hours after onset. B—Same time in Sante's position. C—After bronchoscopic drainage (five hours after onset). D—Two days after bronchoscopy.

PROPHYLAXIS

It seems not too much to predict that the incidence of massive collapse will, in the future, be much reduced even by the most general prophylactic measures. To "think atelectasis" will result, first, in avoidance of too long rest in any one posture. Physiologic atelectasis as found by Webb and by Sewall is too suggestive to be ignored. Soltau[198] says, "To the surgeon the value of placing his patient as soon as possible in the semi upright position will appeal. It is hardly too much to say that all operations on the upper abdomen should be nursed in this position as soon as post-operative shock has passed off. The Fowler frame introduced during the war into casualty clearing stations was an admirable and simple mechanical device for attaining this end." Likewise, the avoidance of bandaging which will embarrass respiration mechanically is wise. On the other hand, abdominal tenderness related to respiration may, of itself, tend to greater fixation than would the bandage which relieves it. A postural technique proposed by Meyer[151] for avoidance of thrombosis is equally applicable to this condition.

To those with time and inclination to

read their paper, the understanding which Legendre and Bailly[123] applied here, and the prophylactic measures they recommend, will form an interesting chapter.

I have elsewhere expressed the opinion that collapse cannot occur in the absence of bronchitis. If this should be only partly true, much attention should be given to the prevention or relief of any manifestation of this condition. Whipple[218] pointedly calls attention to this.

"The patient may easily contract his cold after entering the surgical service. Five patients in this series complained of contracting a 'cold' after admission. These colds may be the result of one of the following factors: The patient usually comes into the hospital too warmly dressed—many of the patients wear more than one suit of heavy underwear; he is given a hot bath and is either put to bed or allowed up and about the cool ward with far less clothing than he is accustomed to wear. During the twelve to twenty-four hours before operation he may be examined in a more or less exposed condition, one or more times. He is given his preparatory shave and is exposed still more in the early morning hours as a result of catharsis and enemas. It may be that coming to or from the operating room he is exposed to cold draughts in the long corridors so characteristic of hospitals. During his recovery from the anesthetic, with his vasomotor system depressed, he may become chilled, especially if his bed clothes do not cover his shoulders; and on the average neatly made hospital bed with blankets shrunken from repeated cleaning, the bed clothes seldom

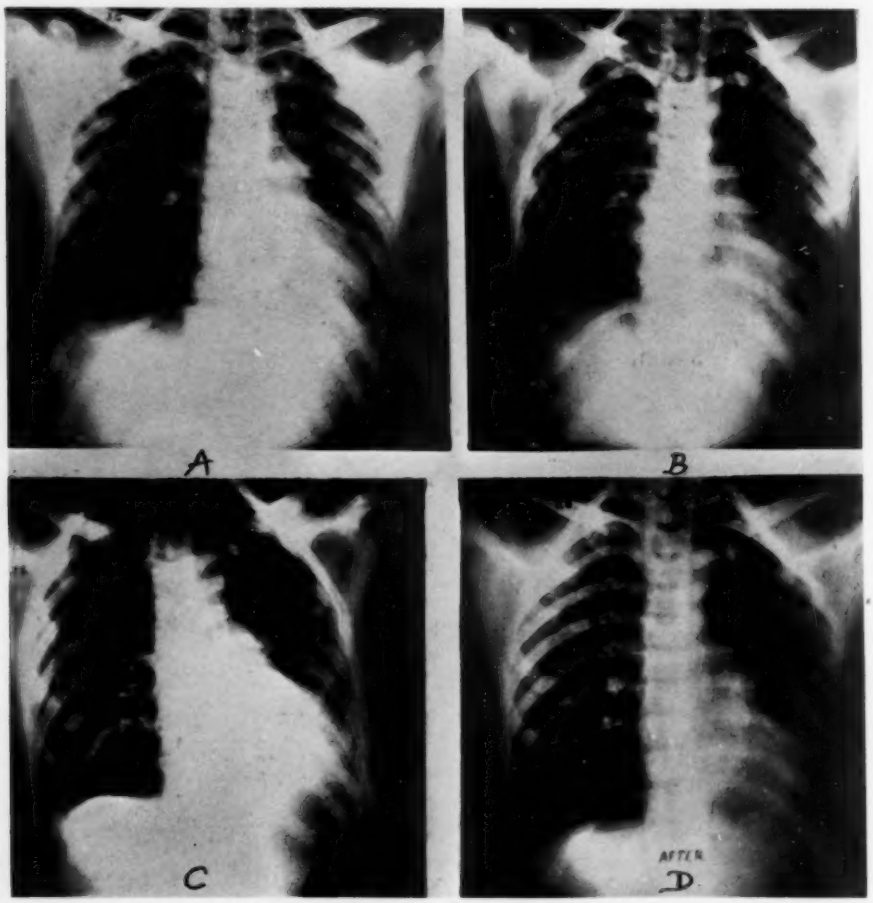


Fig. 10. J. G., male, aged fifteen. Right herniorrhaphy, left lower lobe collapse sixty hours postoperative. A and B—Inspiration and expiration about five hours after onset. C—Inspiration ten hours after onset, immediately before bronchoscopic drainage. D—Immediately after bronchoscopic drainage.

come above the costal margin. A small shoulder blanket may or may not make up for the deficiency. Many of the patients are not accustomed to sleeping in a cold room, and particularly under an open window. Too often the modern intern or nurse with a disdain for

lation of the lungs with carbon-dioxide-oxygen at the end of operation: "This, with a semi-Fowler position after operation, has apparently reduced the incidence of massive collapse as a postoperative complication." Scott and Joelson[186] leave

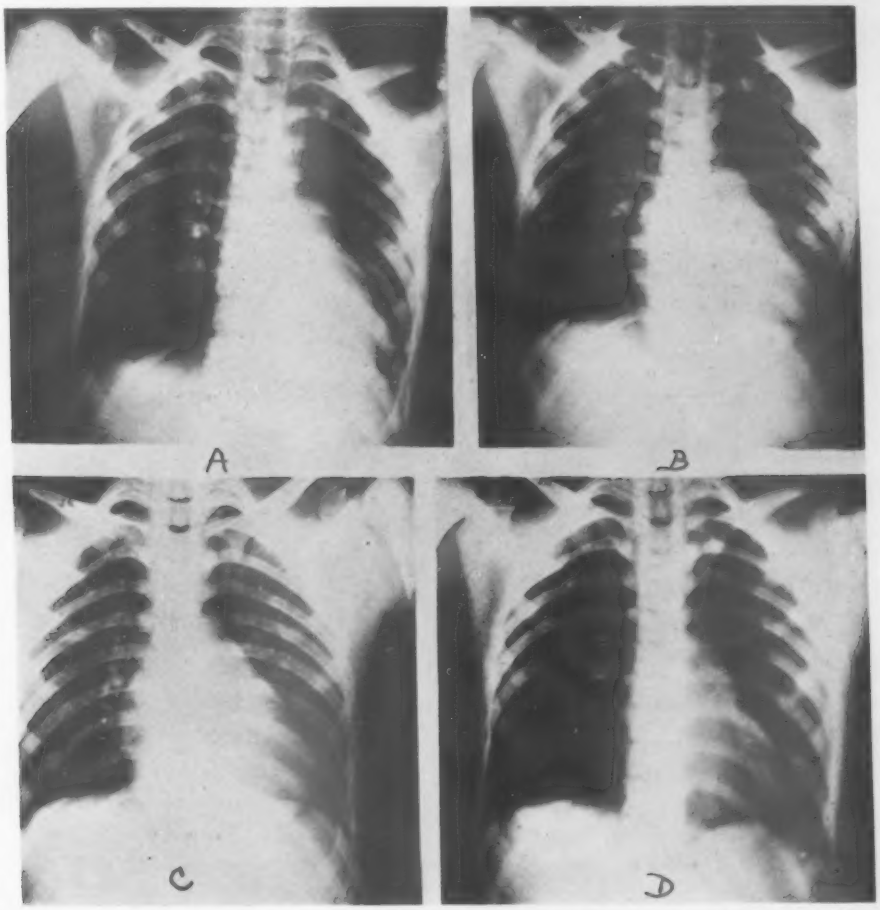


Fig. 11. Same case as in Figure 10. A—Twenty-four hours after drainage, partial relapse. B—Forty-eight hours later, after spontaneous improvement. C—Seventy-two hours. D—Ninety-six hours.

draughts and cool temperature, forgets that the twenty-four hours before and after an operation is not the best time to correct habits and customs of a lifetime. The writer is firmly convinced, from repeated observations in several hospitals, that one of the most important predisposing causes of postoperative pneumonitis both in patients coming into the hospital with a recent cold or free from an active cold, is the exposure to which he is subjected during the first twenty-four to forty-eight hours of his stay in the hospital."

Scott and Cutler[187] propose hyperventi-

little room for doubt that posture is a really deciding factor as to the site of the collapse. They find that if, for instance, the site of the operation leads the patient to choose to lie upon one side continuously, then collapse, if it occurs, will be in the lung restricted by this position. Hunt[95] urges carefully recorded preliminary clinical examination of the chest, supplemented by roentgen examination when possible, for comparison with postoperative findings; hourly change of posture; limitation of morphine before and after operation;

minimum anesthesia and extra postoperative care.

The teaching is obvious. Frequent change of posture is imperative unless forbidden by some definite contraindication.

debility of the patient, untoward result is rare. Recovery is frequently as spectacular as the onset.

TREATMENT

There are no specific remedies and the

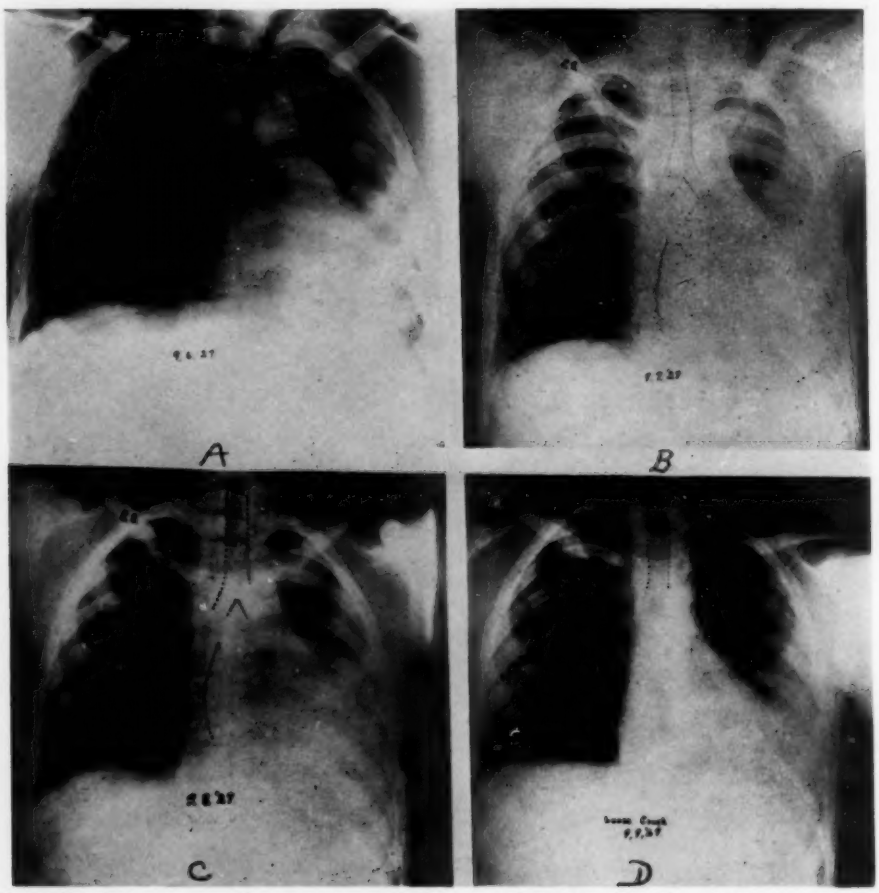


FIG. 12. E. S., male, aged sixteen; emergency appendectomy; collapse left lung fourth day. A, B, C, and D—Second, third, fourth and fifth days, respectively, of collapse.

PROGNOSIS

The gravity of the prognosis is wholly dependent upon the virulence of organisms happening to exist in the collapsed area, and bears little or no relation to the character or degree of the earlier clinical findings. In itself a suspended function, complicating some previous condition, the pathological findings of massive collapse will, like its clinical findings, depend wholly upon the type of infection which it happens to enfold.

Uncomplicated by infection or by serious

use of drugs, if any, must be upon the expectant plan. Mechanically, three measures are proposed.

Elkin[52] says, "It is suggested that the introduction of air into the pleural cavity of these cases showing a marked cyanosis and dyspnoea may be of therapeutic value in bringing the mediastinal structures back to their normal positions." Gammons[64] has so treated one patient, but one having already a tuberculous cavity. Farris[58] treated one tuberculous and one postoperative collapse in this manner. Of

the latter he reports pneumothorax at ten days with return of mediastinum to normal position. "He claimed he felt more comfortable." Film a week later showed the lung clear. Bradford,[21] Mason,[139] Gwyn and others warn against "thorocotomy," in promptly reestablishing aeration of the lung. Jackson and his co-workers have reported reinflation after repeated bronchoscopic removal of mucus from the bronchi. My procedure seems more simple, is available to all, and does not carry any

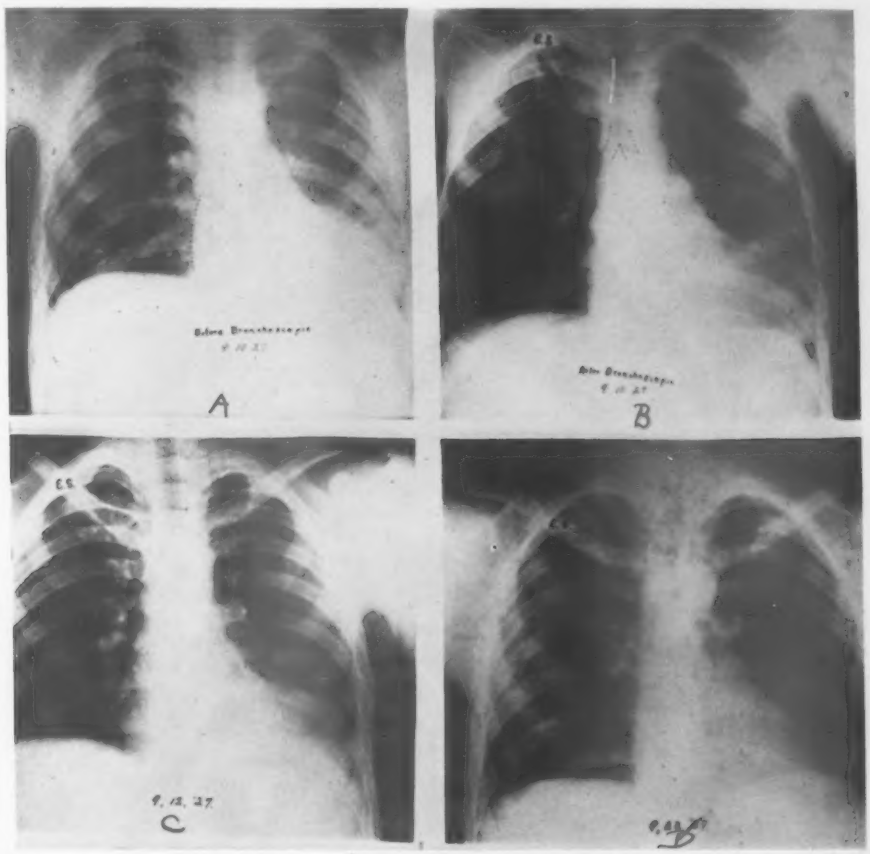


Fig. 13. Same case as in Figure 12. Relapse after partial recovery. A—Sixth day, before bronchoscopy. B—Sixth day, after bronchoscopy. C—Two days after bronchoscopy. D—Twelve days after bronchoscopy; twentieth day of collapse.

"misdirected thoracentesis" and "unfortunate puncturing." Pneumothorax is mentioned here only to condemn it.

Sante,[178] having witnessed a spontaneous reinflation when a patient was turned over on the roentgenoscope, tried rotation in other cases with success and reports the same experience by others. This leads him to state that: "The treatment is simple and consists in rolling the patient back and forth on the uninvolved side. No other therapeutic agent is necessary. This simple procedure has, in all instances in which it has been used, proved successful risk to the patient." This reminds one that Gairdner, some seventy-five years earlier, reported a case in which pneumonia had been suspected but in which he made the diagnosis of collapse. "Under the influence of an emetic and a forced decubitus on the opposite side to that on which the dullness existed, almost every trace of it had disappeared in thirty-six hours, and the dyspnoea was entirely relieved." By this Gairdner proved that the patient had collapse and not "bronchopneumonia." This suggestion of Sante has unquestioned merit. It is logical, harmless, and should

usually be tried, and it may be expected to afford relief in many cases. But we are sure that it will not prove the panacea that he claims. Several careful attempts in our cases quite failed of any satisfactory result. Shellito[192] reports a case in which the patient was found in general collapse shortly after having voluntarily turned to the opposite side. Scott and Cutler[187] comment favorably that "the procedure is so simple that it should be tried in cases of fully developed massive atelectasis. (We must confess that we have not seen any striking results from this procedure in our cases, possibly because we did not continue the process long enough, for when the patient complained of discomfort we allowed him to turn to another position.)" While Sante's method should at least usually be tried it will not always work, and there will be occasional unpleasant experiences with it.

Bronchoscopic drainage, first done by Tucker at the request of Lee, has now 14 successes to its credit and no actual failures. One case, it is true (Hearn and Clerf[78]), was drained seven times but always with relief and finally with cure.

With accumulated experience there will doubtless come improvement. So far drainage has been instituted in the period of active symptoms and as early as possible. Experience may teach that in some instances a delay will mean instituting the drainage at a time when reaccumulation is less likely. It is, of course, true that the greater number would recover with less heroic treatment. The difficulty now, and which promises to continue, is to know whether this present patient is one of that

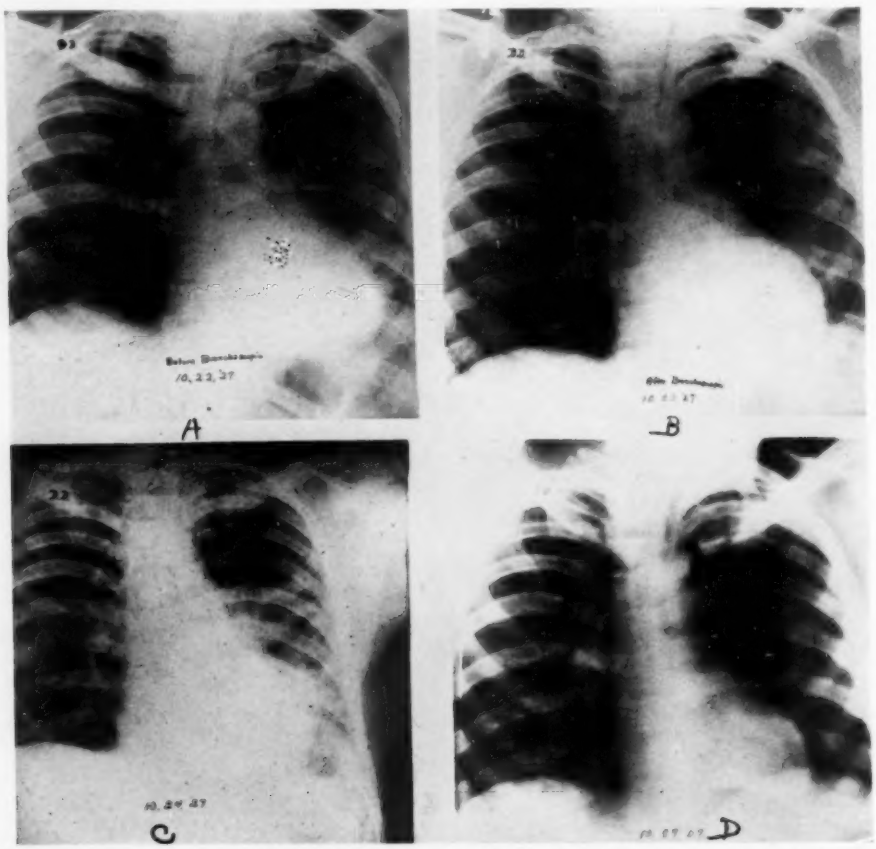


FIG. 14. D. D., male, aged seventeen. Left inguinal herniorrhaphy; left lung collapse forty-eight hours later and bronchoscopic drainage twenty-four hours after onset. A—Before drainage. B—After drainage. C—Two days after bronchoscopy, showing relapse. This was followed six hours later by spontaneous drainage. D—Twelve days after onset.

greater number or whether he is destined for one of the serious, and perhaps fatal, complications.

At the hands of a skillful operator, and the number is rapidly increasing, bronchoscopy is by no means so heroic as it

Sante's method and watchful waiting will be the plan of action.

SUMMARY AND CONCLUSIONS

"This paper exploits no new facts. It is simply an attempt to dust off, rehabili-

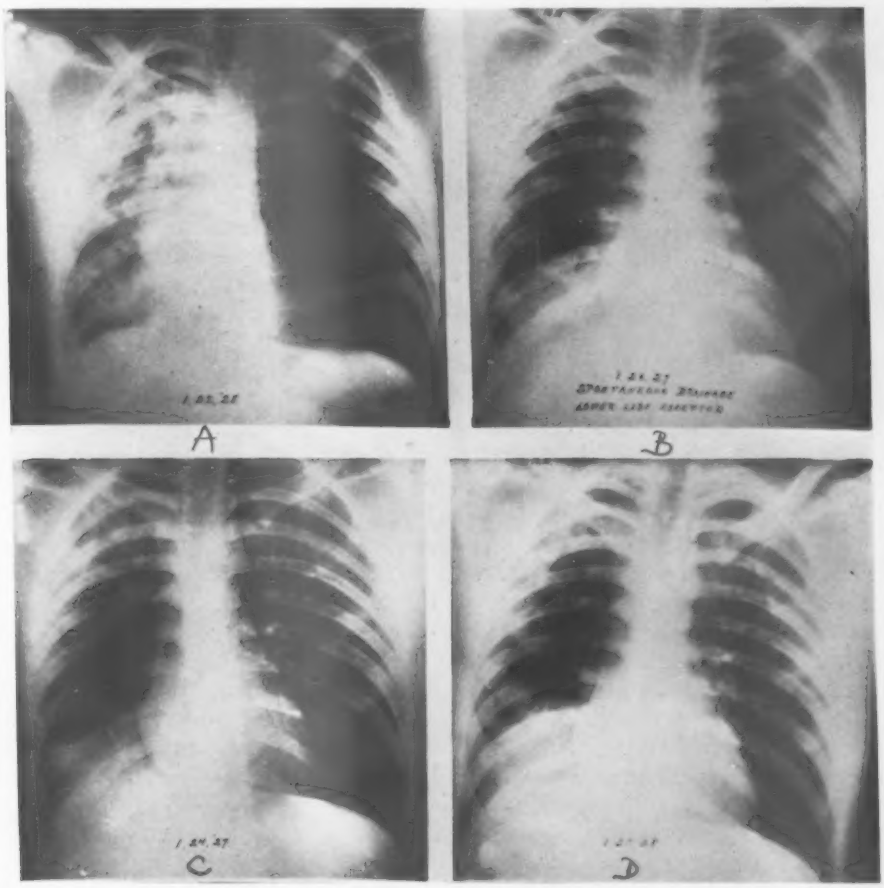


Fig. 15. H. N., male, aged seventeen; appendectomy; collapse right lung forty-eight hours later. A—Typical collapse except that the diaphragm is perhaps not so high as some. B—Spontaneous drainage of upper and middle lobes, second day. C and D—Third and fourth days, no notable change. No further roentgen films were made. Patient discharged well on sixteenth day after operation.

sounds to the uninitiated. Jackson says: "When every surgeon has a bronchoscopic assistant it will be realized that bronchoscopy is as safe and useful as cystoscopy." To one with a roentgenologist's experience with lung abscess, the gross appearance of the 3 to 6 cm. of tenacious, mucopurulent mass which the bronchoscopist delivers is most convincing and heartening. Not less so is the roentgenoscopy of that patient ten minutes later on his way back to the ward.

In the absence of skillful bronchoscopy,

tate and show the value of paraphernalia that most clinicians have cast into a neglected corner" (Sewall).

1. Massive collapse of the lung is a definite clinical entity of which the finally determining etiologic factor is total occlusion of a bronchus with subsequent absorption of retained air. The predisposing and contributing factors of etiology are many.

2. The history and early literature of the subject are fascinating and informative.

3. The principal physical sign is mediastinal displacement toward the involved

lung. While many of the facts were well established in the literature as early as 1850, mediastinal displacement was not reported as an important diagnostic sign until 1911, by Pasteur.

4. The mediastinal, including heart,

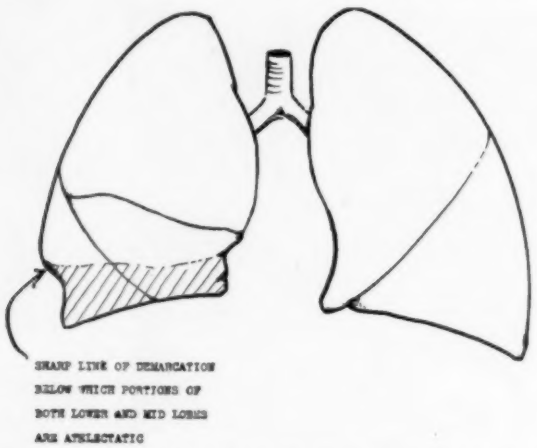


FIG. 16. Sketch by Dr. J. R. Paul depicting typical autopsy findings in one group of the series reported by Lee and Paul. Condition is uniformly bilateral.

displacement is due to the potential thoracic vacuum created by the collapsed lung area, and is directly proportional to the degree in which that vacuum is relieved by emphysema of the opposite lung. Relief of the negative pressure by any other factor, such as elevation of the diaphragm, depression of the chest wall, etc., will, in just the degree in which it is effective, lessen mediastinal displacement.

5. Pasteur was the first to recognize the essential differences separating massive collapse from other postoperative complications, particularly postoperative pneumonia.

6. The postoperative cases, though more noticed recently, are doubtless a much smaller group than those ocurring generally and without traumatism.

7. The striking density of the collapsed lung, by roentgen examination is due to several factors of which vascular engorgement is the most important.

8. The blood supply of the collapsed lung suffers but slight impairment.

9. Any rise of temperature in a postoperative patient after subsidence of reaction should lead the attendant to "think atelectasis," seek roentgen examination, and apply early prophylaxis.

10. Partial, lobular or "patchy" collapse is exceedingly difficult of recognition but nevertheless is an important, in the presence of infection a very important, factor in postoperative prognosis.

11. A high percentage of postoperative lung collapse is preventable by watchful action.

12. The prognosis commonly favorable depends solely upon the type and degree of infection.

13. Without recognition of the early signs at the bedside, the roentgenologist will have scant opportunity to examine these patients. Roentgenology affords the best diagnostic method.

14. Bronchoscopic drainage, skillfully done, is an important method of treatment.

15. When engaged in any phase of lung examination, "think atelectasis."

BIBLIOGRAPHY

1. ABRAMS, A. Observations on physiological pulmonary atelectasis. *Med. Rec.*, 1894, xlvi, 268.

1a. ABT, I. A. Massive collapse of the lung. *Am. J. Dis. Child.*, 1925, xxx, 347–354.

2. ALDERSON, J. On the pathology of hooping cough. *Med.-Chir. Tr., Lond.*, 1830, xvi, 78–93.

3. ANDRUS, W. DE W. Cardiorespiratory physiology following collapse of the lung by bronchial ligation. *Arch. Surg.*, 1925, x, 506–522.

4. ARCHIBALD, E., and BROWN, A. L. Cough; its action on material in tracheobronchial tract; experimental study. *Arch. Surg.*, 1928, xvi, 322–337.

5. ASHBURY, HOWARD. Recurrent massive collapse of the lung due to a benign intrabronchial tumor. AM. J. ROENTGENOL. & RAD. THERAPY, 1929, xxi,

6. ASPER, G. P. Postoperative collapse of lung following nephrectomy. *U. S. Vet. Bur. M. Bull.*, 1927, iii, 1143–1145.

7. AUER, J., and LEWIS, P. A. The physiology of the immediate reaction of anaphylaxis in the guinea-pig. *J. Exper. M.*, 1920, xii, 151–175.

8. BARLING, S. G., and MORRISON, J. T. Manual of War Surgery. Oxford University Press, New York, 1920. (Deals with collapse of penetrated lung only.)

9. BARLOW, G. H. Observations on certain diseases originating in early youth; illustrated by three cases of defective expansion of the lungs. *Guy's Hosp. Rep., Lond.*, 1841, vi, 235–264.

10. BARR, JAMES. The pleurae: Pleural effusion and its treatment. *Brit. M. J.*, 1907, ii, 1289–1295.

11. BARTELS. Bemerkungen über eine im Frühjahre 1860 in der Poliklinik in Kiel beobachtete Masernepidemie, mit besonderer Berücksichti-

gung der dabei vorgekommenen Lungenaffectionen. *Virchow's Arcb. f. patb. Anat. (etc.)*, 1861, xxi, 129–156.

12. Bartels. Beobachtungen über die häutige Bräune. *Deutsches Arcb. f. klin. Med.*, 1867, ii, 367–452.
13. Beals, J. A. Post-operative massive collapse of the lung. *J. Florida M. Ass.*, 1926, xiii, 14–17.
14. Becchini, G. Atelectasis of the lung from foreign body. *Arcb. di radiol.*, 1925, i, 651–653. Abs. in Am. J. Roentgenol. & Rad. Therapy, 1926, xv, 573.
15. Benedetti, P. Il collasso massivo del pulmone (collasso lobare). *Arcb. di pat. e clin. med.*, 1927, vi, 342–367.
16. Bergamini, H., and Shephard, L. A. Bilateral atelectasis (massive collapse) of the lung. *Ann. Surg.*, 1927, lxxxvi, 35–40.
17. Bibergeil, E. Ueber Lungencomplicationen nach Bauchoperationen. *Arcb. f. klin. Cbir.*, 1905, lxxviii, 339–368.
18. Bouchut, J.-E. Handbuch der Kinderkrankheiten, nebst einer Abhandlung über Diatetik und physische Erziehung. Nach der zweiten franzosischen Original aufl. deutsch bearbeitet und reichlich mit Zusatzen versehen von B. Bischoff. Stahel, Wurzburg, 1854.
19. Bowen, C. F. Foreign bodies in the bronchus and esophagus. Am. J. Roentgenol. & Rad. Therapy, 1922, ix, 705–712.
20. Bowen, D. R. Discussion of Lee and Tucker. *Tr. Coll. Phys.*, 1925, xlvii, 246.
21. Bradford, John Rose. Massive collapse of the lung. *Quart. J. Med.*, 1918–19, xii, 127–150.
22. Bradford, John Rose. Massive collapse of the lung. Oxford System Med., 1920–27, ii, 127–136.
23. Brennemann, J. Some observations on the etiology, diagnosis, and treatment of massive collapse of the lung and allied conditions. *Wisconsin M. J.*, 1927, xxvi, 187–189.
24. Briscoe, J. C. The mechanism of post-operative massive collapse in lungs. *Quart. J. Med.*, 1919–20, xiii, 293–336.
25. Brockman, R. St. L. A case of massive post-operative collapse of the lungs. *St. Bartb. Hosp. J., Lond.*, 1913–14, xxi, 63–65.
26. Brown, A. L., and Archibald, E. The action of cough upon material in the tracheobronchial tract; experimental study. *Am. Rev. Tuberc.*, 1927, xvi, 111–122.

26a. Bucher, C. J. The relation between bacterial flora and tracheobronchial foreign bodies. *J. Am. M. Ass.*, 1928, xci, 633–635.

27. Burger, Max. Über die Bedeutung des intrapulmonalen Drucks für den Kreislauf und den Mechanismus des Kollapses bei akuten Anstrengungen. *Klin. Wcbnscbr.*, 1926, v, 825–829.
28. Buzzard, E. F. Clinical history and *post-mortem* examination of five cases of myasthenia gravis. *Brain*, 1905, xxviii, 438–483.
29. Cabot, R. C. Physical Diagnosis of Diseases of the Chest. W. Wood & Co., New York. Third edition, 1905, pp. 359–352; ninth edition, 1927, pp. 351–352.
30. Campbell, H. E. Postoperative massive atelectasis. *China M. J.*, 1927, xli, 711–717.
31. Carlson, A. J., and Luckhardt, A. B. Studies on visceral sensory nervous system. Action of certain drugs on the lung motor mechanisms of the reptilia (turtle, snake). *Am. J. Physiol.*, 1921, lv, 13–20.
32. Chizzola, G. The roentgen picture of atelectasis of the lungs from stenosis of a bronchus. *Arcb. di radiol.*, 1926, iii, 113–121. Abs. in Am. J. Roentgenol. & Rad. Therapy, 1927, xviii, 274.
33. Choyce, C. C. A System of Surgery. Paul B. Hoeber, New York, 1923, iii, 362.†
34. Churchill, E. D. Pulmonary atelectasis, with especial reference to massive collapse of the lung. *Arcb. Surg.*, 1925, xi, 489–518.
35. Churchill, E. D., and Holmes, G. W. Lobar atelectasis in chronic pulmonary suppuration. *Arcb. Surg.*, 1927, xiv, 1093–1106.
36. Clerf, L. H. Seventeen life-saving bronchoscopies in one case. *Surg., Gynec. & Obst.*, 1924, xxxviii, 472–494. (Not significant as to massive collapse except as possible cause for misunderstanding regarding bronchoscopic drainage.)
37. Condie, D. F. A Practical Treatise on Diseases of Children. Blanchard and Lea, Philadelphia, 1858. Fifth edition, pp. 300–306; sixth edition, 1868, pp. 322–328.
38. Conn. J. H. Massive collapse of the lung; post-operative massive atelectasis. *Bull. Univ. Maryland Scbool Med.*, 1928, xii, 223–233.
39. Coplin, W. M. L. Manual of Pathology. Blakiston, Philadelphia, 1911. Fifth edition, pp, 594–595.
40. Corper, H. J., Simon, S., and Rensch, O. B. The effect of artificial pneumothorax on pulmonary tuberculosis in one rabbit. *Am. Rev. Tuberc.*, 1920, iv, 592–600.
41. Coryllos, P. N., and Birnbaum, G. L. Obstructive massive atelectasis of the lung. *Arcb. Surg.* 1928, xvi, 501–559.
42. Coryllos, P. N., and Birnbaum, G. L. Lobar pneumonia considered as a pneumococcic massive atelectasis of the lung. *Bull. N. Y. Acad. Med.*, 1928, iv, 384–399.
43. Crymble, P. T. Gun-shot wounds of the chest. *Brit. J. Surg.*, 1918, v, 363–388.
44. Cutler, E. C., and Hunt, A. M. Postoperative pulmonary complications. *Arcb. Surg.*, 1920, i, 114–157.
45. Cutler, E. C., and Hunt, A. M. Postoperative pulmonary complications. *Arcb. Int. Med.*, 1922, xxix, 449–481.
46. Cutler, E. C. The etiology of post-operative pulmonary complications. *Surg. Clin. N. A.*, 1922, ii, 935–945.
47. Diez, J. El colapso pulmonar masivo post-operatorio. *Prenza med.*, 1927, xiv, 201–220.
48. Dixon, W. E., and Brodie, T. G. Contributions to the physiology of the lungs. Part I. The bronchial muscles, their innervation and the action of drugs upon them. *J. Physiol.*, 1903, xxix, 97–173.
49. Eades, M. F. Postoperative massive atelectasis. *Boston M. & S. J.*, 1926, cxcv, 258–263.
50. Eccles, W. McA. A clinical lecture on gunshot wounds. *St. Bartb. Hosp. J.*, 1914–15, xxii, 69–76.†
51. Edwards, D. J., and Page, I. H. Observations on the circulation during hypoglycemia from large doses of insulin. *Am. J. Physiol.*, 1924, lxix, 177–189.*
52. Elkin, D. C. Intrapleural pressure in post-operative atelectasis. *Ann. Surg.*, 1927, lxxxvi, 885–889.
53. Elliott, T. R., and Dingley, L. A. Massive collapse of the lungs following abdominal operations. *Lancet*, 1914, i, 1305–1309.
54. Elwyn, H. Postoperative pneumonia. *J. Am. M. Ass.*, 1922, lxxix, 2154–2158.
55. Elwyn, H. Postoperative pneumonia. (Second communication.) *J. Am. M. Ass.*, 1924, lxxxii, 384–386.
56. Elwyn, H. A case of acute abscess of lung complicated by massive collapse of the lung. *Boston M. & S. J.*, 1924, cxc, 1108–1111.

57. ELWYN, H., and GIRSDANSKY, J. Postoperative massive collapse of the lung. *J. Am. M. Ass.*, 1922, lxxix, 718–720.
58. FARRIS, H. A. Atelectasis of the lung. *Canad. M. Ass. J.*, 1925, xv, 808–815.
59. FOSTER, S. C. On atelectasis of the lungs; with a case. *Tr. N. York Acad. Med.*, 1851–57, i, 349–365.
60. FUCHS, C. F. Abhandlung über das Emphysem der Lunge. O. Wigand, Leipzig, 1845.
61. FUCHS, C. F. Die Bronchitis der Kinder. O. Wigand, Leipzig, 1849.
62. GAIRDNER, WM. T. On the pathological states of the lung connected with bronchitis and bronchial obstruction. *Month. J. M. Sc.*, 1850, xi, 122; 230: 1851, xii, 440: 1851, xiii, 238.
63. GAIRDNER, WM. T. On collapse of the lung and its results, considered in relation to the diagnosis and treatment of certain diseases of the chest. *Brit. & For. M.-Chir. Rev., Lond.*, 1854, xiii, 207–224.
64. GAMMONS, H. F. Non-pneumatic lung collapse. *Boston M. & S. J.*, 1926, cxciv, 538–539.
65. GARRETT, D. L. Postoperative massive collapse of the lung. *J. Oklaboma M. Ass.*, 1927, xx, 262–264.
66. GMELIN, E. Postoperative Lungenkomplikationen und ihre Bekampfung. *Therap. d. Gegenw.*, 1926, lxvii, 439–441.
67. GOLDEN, ROSS. The effects of bronchostenosis upon the roentgen-ray shadows in carcinoma of the bronchus. AM. J. ROENTGENOL. & RAD. THERAPY, 1925, xiii, 21–30.
68. GOTTSTEIN, G. Erfahrungen über lokale Anästhesie in der Breslauer chirurgischen Klinik. *Arcb. f. klin. Cbir.*, 1898, lvii, 409–434.
69. GRIFFITH, J. P. C. Massive atelectasis (massive collapse) of the lung; with report of case in infant of three weeks. *Med. J. & Rec.*, 1926, cxxiii, 103–106.
70. GRIFFITH, J. P. C. Displacement of the heart in pneumonia in children. *Am. J. M. Sc.*, 1927, clxxiv, 448–453.
71. GWYN, N. B. The clinical side of the condition spoken of as massive collapse of the lung. *Tr. Am. Physicians*, 1923, xxxviii, 410–429.
72. GWYN, N. B. Massive collapse of lungs. *Internat. Clin.*, 1926, i, 136–162.
73. HALL, R. Some rare and obscure pulmonary and pleural conditions. *Lancet*, 1922, i, 61–63.*
74. HALPERIN, G. Postoperative massive collapse of the lung. *Surg., Gynec. & Obst.*, 1925, xli, 53–56. (One case report.)
75. HAMMAN, L. W. Diseases of the lung (atelectasis). Oxford Med., 1920, ii, 65–67.
76. HAMPTON, H. H., and WHARTON, L. R. Venous thrombosis, pulmonary infarction and embolism following gynecological operations. *Johns Hopkins Hosp. Bull.*, 1920, xxxi, 95–116.
77. HARRINGTON, S. W. Relief of post-operative massive collapse of the lung by bronchoscopic aspiration. *Ann. Surg.*, 1927, lxxxv, 152–154.
78. HEARN, W. P., and CLERF, L. H. Post-operative massive collapse of the lung; report of bronchoscopic observations. *Ann. Surg.*, 1927, lxxxv, 54–60.
79. HELLER. Klinische und experimentelle Beiträge zur Kenntnis der akuten Lungenatelektase durch obturierenden Fremdkörperverschluss der Bronchien. *Ztscbr. f. d. ges. exper. Med.*, 1913–14, ii, 453–484.
80. HERB, I. C. Post-operative lung complications. *J. Am. M. Ass.*, 1922, lxxix, 339–342.*
81. HERMANN, L. Das Verhalten das kindlichen bei der Geburt. *Arcb. f. d. ges. Physiol.*, 1882–83, xxx, 276–287.
82. HERTZ. Atelektatische Zustande. Handb. d. spec. Path. (Ziemssen), Leipz., 1874, v, 313–326. Also, transl.: Cycl. Pract. M. (Ziemssen), N. Y., 1875, v, 322–336.
83. HEURER, G. J. Surgery of the thorax. Keen's System of Surgery, 1921, viii, 351.†
84. HEWITT, G. Bronchitis and consequent apneumatosis (pulmonary collapse); with remarks on the diagnosis and treatment of such cases. *Lancet, Lond.*, 1857, i, 625.
85. HEWITT, G. Apneumatosis. System Med. (Reynolds). Lippincott, Philadelphia, 1871, iii, 862–882.
86. HEWLETT, A. W. Diseases of the Lungs. Therapeusis of Internal Diseases (Forscheimer, F.). Appleton, New York, 1917, iii, 418–419.
87. HIRSCH, I. S. The diagnosis of foreign bodies in the alimentary and respiratory tracts of children. *Am. J. Surg.*, 1913, xxvii, 1; 59.
88. HIRSCHBOECK, F. J. Postoperative massive collapse of the lungs. *Am. J. M. Sc.*, 1922, clxiv, 268–274.
89. HIRSCHBOECK, F. J. Massive collapse of lung, with report of necropsy. *Minnesota Med.*, 1928, xi, 135–142.
90. HOFBAUER, L. Therapie der Krankheiten der Respirationsorgane. *Therap. Monatsb.*, 1914, xxviii, 549–555.
91. HOLMES, G. W. Massive collapse of the lung. AM. J. ROENTGENOL. & RAD. THERAPY, 1924, xi, 509–516.
92. HOOVER, C. F. Moisture in the air spaces and oxygen therapy. *Tr. Ass. Am. Physicians*, 1918, xxxiii, 42–54.
93. HOOVER, C. F. Importance of the patient's position in studying lung excursion. *J. Am. M. Ass.*, 1923, lxxxi, 1602–1603.
94. HOWELL, W. H. Text-book of Physiology. Saunders, Philadelphia, 1921. (Chapter on Respiration, p. 632.)
95. HUNT, E. L. Massive atelectasis of the lung as a surgical complication. *Boston M. & S. J.*, 1926, cxciv, 58–65.
96. IGLAUER, S. Tracheo-bronchoscopy, with report of cases. *Obio M. J.*, 1911, vii, 178–183.
97. IGLAUER, S. Three cases of foreign bodies in the bronchi. *Lancet-Clinic*, 1912, cvii, 603–605.
98. IGLAUER, S. Foreign bodies in the bronchi. *Interstate M. J.*, 1917, xxiv, 924–929.
99. JACKSON, C. Anesthesia for peroral endoscopy. *Laryngoscope*, 1912, xxii, 1208–1213.
100. JACKSON, C. The drowning of the patient in his own secretion (editorial). *Laryngoscope*, 1911, xxi, 1183–1185.
101. JACKSON, C. Peroral Endoscopy and Laryngeal Surgery. Laryngoscope Co., St. Louis, 1915.
102. JACKSON, C. Bronchoscopic observations on the cough reflex. *J. Am. M. Ass.*, 1922, lxxix, 1399–1403.
103. JACKSON, C. Postulates on the cough reflex in some of its medical and surgical phases. *Therapeutic Gazette*, 1920, xliv, 609–610.
104. JACKSON, C. Bronchoscopy and Esophagoscopy. Saunders, Philadelphia, 1922, p. 140.
105. JACKSON, C. Bronchoscopy and Esophagoscopy. Second edition. Saunders, Philadelphia, 1927, pp. 158–173.
106. JACKSON, C., SPENCER, W. H., and MANGES, W. F. The diagnosis and localization of non-opaque foreign bodies in the bronchi. AM. J. ROENTGENOL., 1920, vii, 277–285.

107. Jackson, C. Discussion of Lee and Tucker. *Tr. College Physicians*, 1925, xlvii, 247.

108. Jackson, C., Tucker, G., and Clerf, L. H. Arachidic and other forms of vegetal bronchitis. *Atlantic M. Month.*, 1925, xxviii, 506–508.

109. Jackson, C., and Lee, W. E. Acute massive collapse of the lungs, *Ann. Surg.*, 1925, lxxxii, 364–389.

110. Jackson, C., and Lee, W. E. Acute massive collapse of the lungs. *Tr. Am. Surg. Ass.*, 1925, xliii, 723–765.

111. Jenner, W. On typhoid and typhus fevers; an attempt to determine the question of their identity or non-identity, by an analysis of the symptoms, and of the appearances found after death in sixty-six fatal cases of continued fever, observed at the London Fever Hospital from January, 1847, to February, 1849; *Month. J. M. Sc., Lond. & Edinb.*, 1849, ix, 663; 726; 816; 954; 1095; 1264; 1850, x, 106; 308.

111*a*. Jenner, W. On the determining causes of vesicular emphysema of the lung. *Med.-Chir. Tr., Lond.*, 1857, xl, 25–37.

111*b*. Jenner, W. Emphysema of the lungs. Syst. Med. (Reynolds') 1871, iii, 475; 959.

112. Joerg, E. Researches on solidification of the lungs in new-born infants. *Dublin J. M. & Chem. Soc.*, 1834, v, 36–41.

113. Junghagen, S. Some cases of pulmonary atelectasis. *Acta radiol.*, 1926, v, 250–260.

114. Keith, J. P., Bell, J. C., and Keith, D. Y. Massive collapse of the lung. *Kentucky M. J.*, 1927, xxv, 268–273.

115. Klein, F. Massiver atelektatischer Lungenkollaps. *Med. Klin.*, 1928, xxiv, 179–180.

116. Kletz, N. Massive collapse of the lungs complicating case of acute meningitis. *Lancet*, 1927, i, 179–180.

117. Lee, W. E. Post-operative pulmonary complications. *Ann. Surg.*, 1924, lxxix, 506–523.

118. Lee, W. E., and Paul, J. R. Acute massive and partial collapse of the lungs. *Bull. Ayer Clin. Lab. Penn. Hosp., Phila.*, 1926, No. 9, 5–15.

119. Lee, W. E., and Tucker, G. Acute massive collapse of the lungs. *Tr. Coll. Phys. Phila.*, 1925, xlvii, 231–254.

120. Lee, W. E., Tucker, G., and Clerf, L. H. Post-operative pulmonary atelectasis. *Ann. Surg.*, 1928, lxxxviii, 6–14.

121. Lee. W. E., Ravdin, I. S., Tucker, G., and Pendergrass, E. P. Studies on experimental pulmonary atelectasis. *Ann. Surg.*, 1928, lxxxviii, 15–20.

122. Lee, W. E. Postoperative pulmonary atelectasis. *Progressive Med.*, 1927, iv, 165–282.

123. Legendre and Bailly Nouvelles recherches sur quelques maladies du poumon chez les infants. *Arch. gen. de méd., Par.*, 1844, i, 55; 184; 286.

124. Lemon, W. S. Physiologic effect of phrenic neurectomy. *Arch. Surg.*, 1927, xiv, 345–361.

125. Leopold, S. S. Postoperative massive pulmonary collapse and drowned lung. *Am. J. M. Sc.*, 1924, clxvii, 421–433.

126. Lichtheim, L. Versuche über Lungenatelektase. *Arch. f. exper. Path. u. Pharmakol.*, 1878–79, x, 54–100.

127. Lilienthal, H. The relation of radiography to the diagnosis and therapy of non-tuberculous diseases of the lungs and pleura. *Med. Rec.*, 1917, xci, 587–589.

128. Lord, F. T. Diseases of the Bronchi, Lungs and Pleura. Lea and Febiger, Philadelphia, 1915; also second edition, 1924, pp. 133–146.

129. Lord, F. T. Discussion of paper by Gwyn. *Tr. Am. Physicians*, 1923, xxxviii, 428.

130. Louis, P. Ch. A. Recherches de gastro-enterite. C. Baillere, Paris, p. 361.

131. Lund, C. C., and Ritvo, M. Post-operative collapse of the lungs. *Boston M. & S. J.*, 1924, cxc, 1103–1107.

132. Lynch, H. L. Two cases of foreign bodies in the bronchi complicated by general emphysema. *Laryngoscope*, 1915, xxv, 574–577.

133. Mandl, F. Zur Statistik der postoperativen Lungenkomplikationen und über erfolgreiche Bestrebungen zu deren Prophylaxe. *Deutsche Ztschr. f. Chir.*, 1921, clxv, 67–85.

134. Manges, W. F. Roentgen-ray diagnosis of non-opaque foreign bodies in the air passages. Am. J. Roentgenol. & Rad. Therapy, 1922, ix, 288–304.

135. Manges, W. F. Atelectasis as a roentgen-ray sign of foreign body in the air passages. Am. J. Roentgenol. & Rad. Therapy, 1924, xi, 517–523.

136. Manges, W. F. Peanut kernels in the lungs; roentgen-ray diagnosis of non-opaque foreign bodies in air passages. *Surg. Clinics N. Am.*, 1924, iv, 54–65.

137. Manges, W. F. Discussion of paper by Lee and Tucker. *Tr.. Coll. Physicians*, 1925, xlvii, 250.

138. Manges, W. F. Non-opaque foreign bodies in the air passages; x-ray diagnosis and localisation. *Brit. J. Radiol.*, 1926, xxxi, 119–149.

139. Mason, R. L. Massive atelectasis. *Surg. Clinics N. Am.*, 1926, vi, 739–746.

140. Mastics, E. A., Spittler, F. A., and McNamee, E. P. Postoperative pulmonary atelectasis. *Arch. Surg.*, 1927, xv, 155–197.

141. MacCallum, W. G. The teaching of pathological physiology. *Johns Hopkins Hosp. Bull.*, 1908, xix, 215–219.

142. MacCallum, W. G. Textbook of Pathology. Saunders, Philadelphia, 1928, pp. 406–407.

143. McCrae, Thomas. Physical signs of foreign bodies in the bronchi. *Am. J. M. Sc.*, 1920, clix, 313–325.

144. McCrae, Thomas. Diagnosis of a foreign body in a bronchus. *Surg. Clinics N. Am.*, 1924, iv, 16–26.

145. McCrae, Thomas. Clinical features of foreign bodies in the bronchi. *Lancet*, 1924, i, 735–741; 787–793; 838–841.

146. McCrae, Thomas. Discussion of paper by Lee and Tucker. *Tr. College Physicians*, 1925, xlvii, 244.

147. McKinney, J. T., and Porter, W. B. Postoperative massive collapse of the lung. *South. M. J.*, 1927, xx, 681–688.

148. Macleod, J. J. R. Physiology and Biochemistry in Modern Medicine. C. V. Mosby Co., St. Louis, 1920.

149. Meigs, J. F. Remarks on atelectasis pulmonum, or imperfect expansion of the lungs, and collapse of the lungs in children. *Am. J. M. Sc.*, 1852, xxiii, 83–102.

150. Mendelssohn, A. Der Mechanismus der Respiration und Cirkulation, etc. B. Behrs, Berlin, 1845, p. 177 et seq.

151. Meyer, Willy. The importance of posture in postoperative treatment. *Med. J. & Rec.*, 1924 cxix, 590–597.

152. Mikulicz, J. v. Die Methoden der Schmerzbetäubung und ihre gegenseitige Abgrenzung. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, 1901, xxx, 560–593.

153. Mortimer, J. D. Post-graduate lecture on after-effects (and so-called after-effects) of anesthetics. *Med. Press*, 1919, cviii, 505–507.

154. Norris, F. A. Post-operative pulmonary complications. *Illinois M. J.*, 1921, xl, 288–289.

155. Norris, G. W., and Landis, H. R. M. Diseases of

the Chest. Third edition. Saunders, Philadelphia, 1924, pp. 528–532.
156. OTTEN, M. Die Röntgendiagnose der Lungengeschwülste. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1910, xv, 1–31.
157. PASTEUR, W. Respiratory paralysis after diphtheria as a cause of pulmonary complications, with suggestions as to treatment. *Am. J. M. Sc.*, 1890, c, 242–257.
158. PASTEUR, W. Massive collapse of the lung. *Lancet*, 1908, ii, 1351–1355.
159. PASTEUR, W. Active lobar collapse of the lung after abdominal operations. *Lancet*, 1910, ii, 1080–1083.
160. PASTEUR, W. The annual oration on post-operative lung complications. *Lancet*, 1911, i, 1329–1334.
161. PASTEUR, W. Massive collapse of the lung (*Syn.* active lobar collapse). *Brit. J. Surg.*, 1914, i, 587–601.
162. PASTEUR, W. The causation of post-operative massive collapse of the lung. *Lancet*, 1914, i, 1428. (Correspondence concerning paper by Elliott and Dingley, ref. 53.)
163. PEARSON-IRVINE. On a case of diphtheritic paralysis simulating extensive lung disease. *Tr. Clin. Soc., Lond.*, 1876, ix, 188–196.
164. PEPPARD, T. A. Post-pneumonic atelectasis. *Minnesota Med.*, 1927, x, 5–10.
165. PEPPER, O. H. P. Postoperative pulmonary complications, with three illustrative cases. *M. Clinics N. Am.*, 1921, v, 737–749.
166. PFAUNDLER and SCHLOSSMAN. Handbuch der Kinderheilkunde, 1924, iii, 636.
167. PHILLIPS, C. C., and LAFFERTY, R. H. Obstruction of the bronchi by non-opaque foreign bodies. *South. M. J.*, 1923, xvi, 685–688.
167*a*. POWELL, R. D., and HARTLEY, P. H.-S. Diseases of the Lungs and Pleura. H. K. Lewis and Co., Lond., Sixth edition, 1921, pp. 344–351.
168. REGAN, J. C. Massive collapse of the lung in acute poliomyelitis. *Lancet*, 1924, ii, 1222–1224.
169. RIESMAN, D. Diagnostic problems in pneumonia. *J. Am. M. Ass.*, 1924, lxxxii, 1256–1260.*
170. RICHARDS, L. A corn kernel in the bronchus. *Boston M. & S. J.*, 1926, cxciv, 57–58.
171. RIGLER, L. G. Massive collapse of the lung. *Minnesota Med.*, 1926, ix, 326–333.
172. RILLIET, F., and BARTHEZ, E. A Treatise on the Pneumonia of Children (translated from the French by S. Parkman). Carey and Hart, Philadelphia, 1841, p. 15.
173. RILLIET, F., and BARTHEZ, E. Handbuch der Kinderkrankheiten. E. Kollmann, Leipzig, 1855, second edition.
174. RITVO, MAX. Massive collapse of the lung. AM. J. ROENTGENOL. & RAD. THERAPY, 1924, xi, 337–342.
175. RUPP, A. Postoperative Thrombose und Lungenembolie. *Arch. f. klin. Chir.*, 1921, cxv, 672–688.
176. SANTE, L. R. Acute consolidations of the lung; their recognition and differential diagnosis. *Radiology*, 1925, iv, 221–241.
177. SANTE, L. R. Massive collapse of the lung. *Radiology*, 1927, viii, 1–13.
178. SANTE, L. R. Massive collapse of the lung. *J. Am. M. Ass.*, 1927, lxxxviii, 1539–1542.
179. SANTE, L. R. Massive (atelectatic) collapse of the lung. *Ann. Surg.*, 1928, lxxxviii, 161–177.
180. SANTEE, H. E. Bilateral collapse of the lungs. *Ann. Surg.*, 1927, lxxxv, 608–609.
181. SAUERBRUCH, F. Chirurgie der Brustorgane. J. Springer, Berlin, 1920, i, 172–175.
182. SCHIASSI, F. Il collasso massivo del pulmone. *Arch. di pat. e. clin. med.*, 1924, iii, 553–573.
183. SCHOLTZ, C. J. Massive collapse of the lung. *Med. J. S. Africa*, 1921, xvi, 202–205.
184. SCHROEDER, W. E., and GREEN, F. R. Phrenic nerve injuries. *Am. J. M. Sc.*, 1902, cxxiii, 196–220.
185. SCOTT, W. J. M. Postoperative massive collapse of the lung. *Arch. Surg.*, 1925, x, 73–116.
186. SCOTT, W. J. M., and JOELSON, J. J. Postoperative massive atelectasis; influence of posture. *Arch. Surg.*, 1927, xv, 855–870.
187. SCOTT, W. J. M., and CUTLER, E. C. Postoperative massive atelectasis; effect of hyperventilation with carbon dioxide. *J. Am. M. Ass.*, 1928, xc, 1759–1763.
188. SCRIMGER, F. A. C. Postoperative massive collapse of the lung. *Surg., Gynec. & Obst.*, 1921, xxxii, 486–492.
189. SCRIMGER, F. A. C. Postoperative massive collapse of the lung. *Am. J. Surg.* (*anesthesia suppl.*), 1922, xxxvi, 50–53.
190. SEWALL, HENRY. Pulmonary atelectasis as a source of confusion in physical examination of the chest. *Am. Rev. Tuberc.*, 1921, iv, 811–827.
191. SEWALL, HENRY. A critique on artificial pneumothorax in pulmonary tuberculosis; with special reference to the mobility of the mediastinum. *Am. Rev. Tuberc.*, 1928, xviii, 117–201.
192. SHELLITO, J. C. Post-operative massive collapse of the lung. *J. Iowa M. Soc.*, 1926, xvi, 186–188.
193. SHONLE, H. A., and MOMENT, A. Some new hypnotics of the barbituric acid series. *J. Am. Chem. Soc.*, 1923, xlv, 243–249.
194. SHIPWAY, F. E. Post-operative pulmonary affections. *Lancet*, 1925, i, 409.
195. SIGNORELLI, A. Criteri obbiettivi di cervezza per la diagnosi di broncopneumorragia. *Radiol. med.*, 1917, iv, 300.
196. SMITH, F. J., and DAVIDSON, E. C. Post-operative pulmonary atelectasis. *J. Michigan M. Soc.*, 1927, xxvi, 295–300.
197. SOLEM, G. O. Massive collapse of the lung. *M. Clin. N. A.*, 1927, xi, 105–112.
198. SOLTAU, A. B. Massive pulmonary collapse. *Brit. M. J.*, 1925, i, 544–547.
199. SPEISS, G. Ein fall hochgradiger Dyspnoe infolge eines Polypen im rechten Bronchus. *München. med. Wchnschr.*, 1910, lvii, 2095–2096.
200. STOKES, WM. A Treatise on the Diagnosis and Treatment of Diseases of the Chest. Hodges and Smith, Dublin, 1837, pp. 189–192.
201. TERRY, R. J. On the presence of fluid in the pulmonary alveoli. *J. Missouri M. Ass.*, 1920, xvii, 40–41.
202. THOENES, F. Ueber Mediastinalverlagerung bei infiltrativen Lungenerkrankungen im Säuglingsalter. *Monatschr. f. Kinderh.*, 1922, xxiii, 353–365.
203. THORNTON, W. L., and PRATT, J. P. The relation of bronchial stenosis to bronchiectasis. *Johns Hopkins Hosp. Bull.*, 1908, xix, 230–232.
204. TIDY, H. L., and PHILLIPS, E. Acute lobar collapse of the lung. *Lancet*, 1914, i, 1245–1247.
205. TRAUBE, L. *Beitr. z. exper. Path. u. Physiol.*, 1846, i, 184 et seq.
206. TRAUBE, L. *Ges. Beitr. z. Path. u. Physiol.*, 1871, i, 1–112.
207. TROUT, H. H., and HAYTER, H. M. Massive collapse of the lung. *Virginia M. Month.*, 1926, liii, 214–221.
208. TUCKER, G. Recent developments in peroral endoscopy. *Surg., Gynec. & Obst.*, 1926, xlii, 743–752.
209. VIERORDT, K. H. Anatomische, physiologische und physikalische Daten und Tabellen. Third edition. G. Fischer, Jena, 1906.

210. WALLGREN, A. Displacement of the heart in pneumonia. *Acta paediat.*, 1923, iii, 81–90.
211. WEBB, G. B., FORSTER, A. M., and HOUCK, F. M. Rest of the individual lung by posture. *J. Am. M. Ass.*, 1916, lxvii, 1422–1423.
212. WEBB, G. B., FORSTER, A. M., and GILBERT, G. B. Postural rest for tuberculosis. *J. Am. M. Ass.*, 1921, lxxvi, 846–849.
213. WEIL, A. Handbuch und Atlas der topographischen Percussion. Second edition, F. C. W. Vogel, Leipzig, 1880.
214. WEINBERGER, M. Linkseitige Bronchostenose durch Aortenaneurysma und Retraktion des Herzens sowie des Mediastinums in die linke Thoraxhälfte. *Mitt. d. Gesellsch. f. inn. Med. u. Kinderh. in Wien*, 1906, v, 204–208.
215. WESSLER, H., and JACHES, L. Clinical Roentgenology of Diseases of the Chest. Southworth Co., Troy, N. Y., 1923. See pp. 48, 49 and 289.
216. WEST, CHARLES. Lectures on the Diseases of Infancy and Childhood. Longman, London, 1848; also Lee and Blanchard, Philadelphia, 1850.
217. WEST, SAMUEL. Plastic bronchitis; extreme displacement of the heart and mediastinum produced by collapse of the lung. *Lancet*, 1908, i, 489–490.
218. WHIPPLE, A. O. A study of post-operative pneumonitis. *Surg., Gynec. & Obst.*, 1918, xxvi, 29–47.
219. WILLSHIRE, W. H. Historic data, etc., in reference to some points of infantile pathology. No. 1. Pneumonia. *Brit. & For. M.-Chir. Rev., Lond.*, 1853, xii, 514–529.
220. YATES, J. L. Effects of acute and chronic pneumothorax. *Am. J. M. Sc.*, 1923, clxv, 1–11.

* Listed because it appears in other bibliographies. Its relation to this subject is remote or even lacking.
† Contains references to pulmonary massive collapse but they are too brief or too generalized to carry practical information.

A FURTHER STUDY OF THE ADVISABILITY OF A GENERAL ROENTGENOLOGICAL CHEST SURVEY APPLIED TO RECRUITS OF THE UNITED STATES ARMY*

BY CHARLES L. MAXWELL, M.D.

Captain, Medical Corps, U. S. Army

Roentgenologist, General Dispensary

WASHINGTON, D. C.

THIS is the second of a series of papers dealing with the tuberculosis problem of the United States Army, and its purpose is to attempt to show the reasons for and the advantages which may accrue from a general roentgenological chest survey of recruits or applicants for enlistment.

In 1926 a survey was made of the cases of pulmonary tuberculosis found in the Hawaiian Department, United States Army, for the years 1923, 1924, and 1925. The summary revealed that 80 per cent of these cases were in first enlistment men with less than three years' service and that 39 per cent had less than one year's service, at the time they were diagnosed as tuberculous.[1]

The degree and type of involvement in practically all of these cases showed plainly that most of them were tuberculous at the time they entered the Service, and that the condition was not discovered by the existing methods of examination.

The natural deduction was made that this influx of undesirables could be prevented by routine roentgen surveys of the chest.

The marked degree of involvement in the vast majority of these cases has been so great that I have searched for a way to place the subject before the Medical Service so that a fuller realization of the situation might be had.

With that in mind the cases of pulmonary tuberculosis, occurring in soldiers having less than one year's service, and who were admitted to Fitzsimons General Hospital, Denver, Colorado, during the years 1925, 1926, and 1927, are taken, and from these a number selected for lantern slide demonstration.

The number of cases for this period is

[1] Maxwell, C. L. Advantages of a roentgenological chest survey applied to recruits of the United States Army. AM. J. ROENTGENOL. & RAD. THERAPY, 1927, xviii, 562–564.

* Published with permission of the Surgeon General, U. S. Army, who is not responsible for any opinion expressed or conclusions reached herein. Read at the Twenty-ninth Annual Meeting, AMERICAN ROENTGEN RAY SOCIETY, Kansas City, Mo., Sept. 25–28, 1928.

sixty-four. The total number of original enlistments for the same period is 102,000. This gives a rate of 0.06 per thousand, of recruits who were manifestly tuberculous at the time of induction into the Service and who were regarded on enlistment, following the usual physical examination, as physically fit.

A study of these cases with regard to the amount of involvement is shown in the following:

	Number of Cases	Per Cent
Involvement of both lungs	46	72.0
Involvement of more than one lobe, one lung, with the opposite lung clear	10	15.5
Involvement of only one lobe	8	12.5

The very high percentage of well-advanced tuberculosis is at once apparent and the hopelessness of most of them, so far as ever regaining health is concerned, is readily appreciated.

An effort is made to estimate the amount of fibrosis by using the terms slight and marked.

	Number of Cases	Per Cent
Cases showing slight fibrosis	17	26.5
Cases showing marked fibrosis	47	73.5

In using the term fibrosis the generalized fibrosis of the lungs often seen is taken with those cases showing frequently a localized fibrosis of one part of the lung and rapid infiltration of other parts with little or no fibrosis. The purpose of mentioning the fibrosis is to more definitely make certain that the tuberculous lesion in some part of the lung has existed for a much longer time than a year.

The next comparison is made with regard to cavitation, whether single or multiple and large or small. The results follow:

	Number	Per Cent
Cases with no cavitation	17	26.5
Cases with cavitation	47	73.5

	Number	Per Cent
(*a*)		
Cases with single cavitation	11	17.0
Cases with multiple cavitation	36	56.5
(*b*)		
Cases with large cavities	14	22.0
Cases with small cavities	33	52.0

It is to be noted that the cases showing multiple cavitation are almost entirely of the small cavity type, and that only two cases in the series showed multiple large cavitation. Again the gravity of most of these cases is realized when the percentage showing cavitation reaches almost 75 per cent.

Lower lobe pathology predominated, either as a true infiltration or as a pleurisy in 4 cases, and 10 cases showed a marked pleural involvement of some part of the lung. All those showing a predominating lower lobe lesion also had some type of upper lobe abnormality of an older nature.

In this series of 64 cases it is believed that but few should have been passed by the average clinical examiner, and since the training which Army medical officers are given is at least average, the deduction may be made that here at least are cases, usually considered easy to discern under the ordinary standard ways of examination that are most elusive, and may it also be said that perhaps the evidence will warrant the assumption that the present methods of examination might well be more often aided by such a positive aid as a roentgen survey of the chest.

The estimated cost to the Government at the present time to enlist, maintain and discharge the average tuberculous patient is $1500.00 and this gives a total of $96,000 as the cost of enlisting, maintaining and discharging those found tuberculous, at Fitzsimons General Hospital, with less than one year's service for the period 1925, 1926, and 1927.

Using the deduction of the investigation made into the tuberculosis situation in the Hawaiian Department one may expect 165 tuberculous patients to reach the hospital from the 112,000 recruits enlisted during this period, and that at least 90 per cent of these will show evidence of a tuberculosis that would have caused their rejection at the time of enlistment. The estimated cost for this group is about $200,000 or at least double the cost of examining all recruits before enlistment.

Practically all Army hospitals have equipment to make routine roentgen examination and the larger centers where most of the recruiting ends or where most

recruits drift to are amply supplied with the necessary equipment.

The post-graduate training given the Army medical officers at the Army Medical School peculiarly adapts them for this type of examination and there are but few who cannot readily adapt themselves to this branch of work.

Again at all the larger centers there is a specially trained personnel and in at least one post in every corps area there is a specialist maintained who is within easy call to aid in doubtful decisions.

To most of you, this record of cases not recognized in the usual clinical examination is an old, old story, but in approaching the subject from this angle the repetition may be worth while in that only by the use of such a volume of the routine side of chest roentgenography can a marked impression of the situation be made.

Especially do I want to interest those of you who are in the Medical Reserve Corps in the furthering of this problem and also to sow a small seed among those who are interested in large commercial units. Their interests and the Army's from this standpoint are almost identical and a survey such as suggested, is as feasible and as proper a procedure in large commercial units as in the military service, and will yield equal results.

The deductions drawn from this series of cases are as follows:

1. That too many cases of well-developed pulmonary tuberculosis are being passed by medical examiners at enlistments for the United States Army under the present methods of examination.

2. That this undesirable element is an exceptional menace to those about them because of the fact that they are accepted as physically fit, when at least 80 per cent of the tuberculous cases are expectorating tubercle bacilli at the time they enter the Service.

3. That at least 90 per cent of these cases could have been discovered by a roentgen chest survey at enlistment and prevented from entering the Army.

4. That this examination would be a real step in economy since the cost of keeping these cases out of the Army would be less than the cost of maintaining and discharging them later.

5. That the necessary examinations can be successfully carried out at most posts of the United States Army without the addition of further equipment and that the training of the majority of medical officers fits them well for such work.

MASSIVE HEMORRHAGE FROM THE GASTROINTESTINAL TRACT*

BY L. R. SANTE, M.D.

ST. LOUIS, MISSOURI

FOR many years cases have been encountered where massive gastric hemorrhages have occurred without apparent cause. Before the days of roentgenology such cases were usually ascribed to esophageal varices, gastric or duodenal ulcer or cancer. The perfection attained by roentgenologists in recent years in the diagnosis of lesions of the gastrointestinal tract has led to a more critical examination of such patients.

As early as 1803 Dalché[5] mentioned the possibility of hemorrhage from the stomach without organic lesion, and in 1815 Latour[11] published a book in Orleans, in which he recognized hemorrhage "*sine materia*," and quoted cases from Portal, Franck, Hoffmann, van Swieten, Baldinger and Warton.

In 1906 White[19] collected 29 cases of massive hemorrhage, selecting only those proved by autopsy or operation to be free from organic lesion. At the suggestion of Sir Edwin Cooper Perry he applied the name "gastrostaxis."

In 1914, Deaver[6] published a paper on gastric hemorrhage in which he ascribed chronic focal infection, either local or remote, as a cause for such mysterious hemorrhages: "Excepting extrinsic poison, I believe the violent congestion of the gastric vessels is primarily dependent on an intra-abdominal or more rarely remote infection." This focus of infection he believed was most commonly located in the gall-bladder or appendix.

Rosenow,[17] in 1915, showed the association of various streptococcus infections with bleeding from hemorrhagic points and superficial ulcers of the stomach.

In the same year Eusterman[8] pointed out that gastric hemorrhage without local lesion was associated with infection of the gall-bladder and gastric symptoms in 5 per cent, and with appendicitis in 2 per cent of all cases at the Mayo Clinic in the series studied.

In 1916, Moschcowitz[13] published 4 cases in which massive hemorrhage had occurred, but in which no demonstrable local hemorrhage could be detected at operation.

In the same year Smithies and Bowen[18] described 5 cases of vicarious hemorrhage from the stomach at the menstrual period in which extensive surgical exploration of the stomach failed to reveal any cause or to give any relief.

Crispin,[4] in 1917, stressed the toxic factor as a cause for the condition and stated that with infections of the gall-bladder and appendix there are occasional hemorrhages for which no adequate cause in the stomach is found at operation. He suggested the term "toxic gastric hemorrhage."

Outland and Clendening,[15] in 1917 describe in detail the operative findings in 3 cases of gastric hemorrhage. "The gall-bladder was empty of stones, with thin walls, and easily emptied by pressure. The stomach had no sign of ulcer, no scar, no dimpling, and the pylorus was patent. The surface of the gastric serosa around the pylorus was reddened and mottled with small veins like a drunkard's cheek. The veins in the region were distended and tortuous, and in the mesentery along the greater curvature were numerous small, hard, shot-like glands.

"On investigating the appendix it was found buried in a dense mass of adhesions, from which it was removed with great difficulty" . . . They ascribe the distortion of the blood vessels to a stream of infection carried up the omentum to the stomach and are of the opinion that the hemorrhage from the stomach was almost certainly due to seepage from the inflamed gastric mucosa.

In 1918, Drybrough-Smith[7] called attention to hematemesis as a postoperative complication following appendectomy, and suggested that many of these cases may have atoxic origin and not be due to any technical operative fault.

* From the Department of Radiology of St. Mary's Hospital and St. Louis City Hospital, St. Louis, Mo. Read at the Twenty-ninth Annual Meeting, AMERICAN ROENTGEN RAY SOCIETY, Kansas City, Mo., Sept. 25–28, 1928.

Nicolaysen,[14] in 1920, suggested the possible cause as of nervous origin and pointed out the effect of the vegetative nervous system on the stomach. He cites the experiments of Finzi[9] who removed various portions of one or both suprarenal glands in rabbits and dogs, and subsequently observed edema and hemorrhage in the gastric mucosa, degeneration of the epithelial cells and even real ulceration; the greater the amount of suprarenal removed, the greater the effect.

Monod,[12] in 1924, reviewed the literature and described similar findings in 3 female patients.

In the same year Bevan[3] analyzed the common causes of gastric hemorrhage and called attention to gastric hemorrhage after operation on abdominal organs. He quotes Eiselberg as regarding such postoperative gastric hemorrhage as thrombosis of the abdominal veins. In another article, Friedenwald and Wiest[10] enumerate the causes of massive gastric hemorrhage and call attention to the possibility of sudden rupture of arteriosclerotic gastric vessels as a possible cause.

It will be seen from a consideration of reported cases that there is substantial basis for the assumption that gastric hemorrhages occur at times under very obscure conditions. Consider the common causes of gastric hemorrhage as outlined by Friedenwald and Wiest:

1. Gastric or duodenal ulcer.
2. Carcinoma.
3. Exulceratio simplex (Dieulafoy).
4. Hemorrhagic gastritis (including alcoholic gastritis).
5. Hepatic affections (cirrhosis with esophageal varices, epidemic jaundice, acute yellow atrophy).
6. Splenic diseases (anemia, Banti's disease).
7. Constitutional and toxic diseases (leucemia, hemophilia, yellow fever, phosphorus poisoning).
8. Aortic aneurysm with rupture into the stomach.
9. Trauma over the stomach.
10. Vicarious menstruation.
11. Localized arteriosclerosis.

A close analysis of these causes will show that they fall into three more or less well-defined classes:

A. Organic lesions, which by their destruction of tissue erode blood vessels, such as gastric ulcer and cancer.

B. Mechanical conditions, such as trauma, rupture of aneurysm, arteriosclerotic or varicosed vessel.

C. Toxic states, local or general.

The first two causes do not require further consideration since they are quite commonly encountered, and their pathological physiology fully understood. The exact manner in which toxemia operates in the production of gastric hemorrhage is not definitely known, yet it is generally believed that the toxin exerts a deleterious effect on the constituents of the blood and capillary walls causing them to become permeable and permitting the passage of blood. It is possible that the effect in these cases is wholly or for the most part due to changes in the character of the blood.

Hemorrhage occurs in epidemic jaundice, acute yellow atrophy of the liver, splenic anemias, Banti's disease, lymphatic leucemia, yellow fever, malaria, phosphorus poisoning, etc. All of these conditions produce profound states of intoxication, or present other grave symptoms of disease.

Massive hemorrhages occur, however, under other circumstances in which there is no evidence of constitutional disease, and frequently where there are few symptoms of any localized disorder. The cases of this type most frequently encountered usually give a history of periodic attacks of indigestion, belching of gas or bloated feeling after eating, extending over a period of years. Suddenly without apparent cause, during one of these exacerbations, the epigastric pain becomes intense, they become nauseated and vomit quantities of blood. Such hemorrhagic attacks may recur at frequent intervals; one patient who has come under the writer's observation was never free from such an attack of gastric hemorrhage for longer than six weeks, for over four years; the hemoglobin fell as low as 12 per cent. Further investigation or operation usually discloses chronic infection of the gall-bladder or appendix, or some structure which empties into the intestinal tract, and removal of the diseased organ usually removes the

tendency to hemorrhage. The following case is a very interesting example:

W. J. R., white, male, aged fifty-two, entered the hospital Dec. 31, 1921, with a history of epigastric pain, nausea and vomiting of coffee ground material. Nothing in the previous history was of significance other than the fact that he had been subject to mild attacks of indigestion for the previous twenty years. Repeated gastrointestinal examinations failed to reveal any evidence of organic lesion other than diverticulitis of the entire large bowel. Several hemorrhages were recorded during the next three weeks but after that the disturbance subsided and the patient recovered to such an extent that he was discharged on Feb. 4, 1922. Gastric analyses showed normal hydrochloric acid content, and periodically tests for blood were positive both from the vomitus and stool. The red blood count at one time, following a severe hemorrhage, fell to 1,030,000 and the hemoglobin to 20 per cent; white blood count ranged from 12,000 to 14,000; blood Wassermann was negative.

The patient was readmitted about two weeks later, Feb. 17, 1922, complaining of a recurrent attack of abdominal pain and vomiting of a large quantity of blood. Within four days after admission, the patient was submitted for gastrointestinal examination without alkalinization or any other medication for his gastric condition. No organic lesion was found other than diverticulitis of the colon. Following an extremely severe hemorrhage the patient died, April 24, 1922. At autopsy the entire gastrointestinal tract was found filled with blood, small and large bowel as well as the stomach. There was no ulcer or cancer of the stomach or other organic lesion of the gastrointestinal tract other than multiple diverticula of the colon.

Robineau[16] has reported a similar case associated with diverticulum of the fourth portion of the duodenum.

Still another case having very important features is that of Mrs. C. S., white, aged forty-seven (patient of Dr. S. I. Frankel, to whom I am indebted for this report). Without premonitory symptoms the patient was seized the evening before with violent abdominal pain followed shortly after with extensive urticarial eruption. About one-half hour later she became nauseated and vomited a large quantity of dark blood, probably exceeding a quart, according to the physician's notation. She gave a history of periodic attacks of indigestion, but never as severe as this and never associated with hematemesis. She was admitted to the hospital Feb. 13, 1928, for examination. Roentgenographic examination failed to reveal any evidence of organic lesion, the patient recovered completely within a week or so, and has had no return of the trouble since.

It is difficult to determine accurately the percentage of instances in which hemorrhage from the gastrointestinal tract is unassociated with organic lesion since most of these cases having no pathology are catalogued as negative. A conservative estimate, however, would probably show not less than 1 per cent of all cases of hematemesis to be without organic lesion.

In seeking an explanation of the manner in which focal infection in the gall-bladder, appendix, or other organ could cause hemorrhage from the stomach or intestines, it has always seemed to us that an explanation merely on the basis of dilated blood vessels is not entirely satisfactory. The experimental work of Auer[2] in producing gastric hemorrhage as a result of anaphylactic reaction in guinea pigs, rabbits and dogs, suggests that anaphylaxis may be the basis of a similar reaction in man. One of the cases cited in which hemorrhage was preceded by a general urticarial reaction lends credence to this view. Auer has shown that hemorrhage occurs from the stomach in guinea pigs previously sensitized to horse serum, when an anaphylactic reaction is produced by further injection of the sensitizing agent. His description of the gastric lesions found at autopsy is as follows:

"The earliest lesions observed consisted of short, superficial linear or starshaped, brownish erosions of the mucosa in the middle third of the stomach; they were located on both anterior and posterior surfaces, and generally much nearer the greater curvature than the lesser one. Careful inspection showed no sign of blood either in the stomach contents or in the erosions themselves.

"In the next stage hemorrhages of peculiar structure and variable extent were noted. In typical instances the hemorrhage was composed of small, flattened, black, discoid masses of blood more or less superimposed one upon the other so that sometimes the hemorrhage looked like a miniature compressed bunch of grapes. On removal of the hemorrhages by washing in Ringer solution, linear irregular erosions were disclosed; these were always

fewer in number and smaller in extent than was expected from the size of the hemorrhage . . .

"Local extreme dilatation of many but not all mucosal blood capillaries was very common in the middle third of the stomach, but was not generally observed to the same degree in the preantral and antral regions. These local capillary dilatations were always especially noticeable near areas of hemorrhage . . . The arteries and veins in the submucosa showed markedly dilated and constricted sections, so that they looked irregularly beaded."

He further explains the findings as follows:

" . . . the erosions and hemorrhages are the result of a local auto-digestion. This auto-digestion is localized because it only occurs in asphyctic areas of the mucosa. The local asphyxia is caused by a decrease or stoppage of the blood circulation in the capillaries of that region. The focal capillary circulation is deficient because apparently travelling peristaltoid contractions in the arterioles, veins and even capillaries impede or temporarily abolish the transit of blood in these areas. These peristaltoid contractions also would explain the peculiar structure of the hemorrhages alluded to above" . . .

It is therefore at least theoretically possible that a severe anaphylactic reaction may explain gastric hemorrhage unassociated with organic lesions in man. The fact that in the greatest majority of instances hemorrhages of this type are associated with chronic infection of the gall-bladder, appendix or diverticula of the intestines may indicate their association as a *localized anaphylactic phenomenon.* Further work of Auer[1] tends to show that if an individual sensitized to any foreign protein is again subjected to a subliminal dose of the same agent, insufficient in itself to produce generalized reaction, a localized anaphylactic reaction will occur at any site of inflammation or injury at that time present in the body or produced soon after while the agent is still circulating in the blood. To quote Auer's experimental work: Rabbits sensitized against horse serum, were again inoculated with a subliminal dose of horse serum and a few minutes later a local irritant (xylol) was applied to the rabbit's ear. Hemorrhagic reaction occurred and dry gangrene resulted in loss of as much as half of the end of the ear, a reaction which was not present in any of the normal, or sensitized rabbits used as controls. This reaction is interpreted as being the result of local antoinoculation in a sensitized rabbit, the sensitizing agent, circulating in the blood is carried in excess to a single locality (ear) by reason of the local inflammation and congestion caused by application of an irritant. A subliminal concentration of antigen for non-inflamed sensitized cells may thus become effective when inflamed sensitized cells are concerned. This process may theoretically occur in any tissue of a sensitized animal which can show an anaphylactic reaction, for example, the intestines, lungs, heart, skin, nerves, arteries, etc. It is possible that this interplay of conditions may explain a number of functional abnormalities in the human subject. It is theoretically possible therefore, that chronic infection of the gall-bladder, appendix, or diverticulum of the intestinal tract, may produce sensitization in the individual to these products of bacterial growth; that such a sensitized individual may, by the intermittent dumping of the same infectious material into the intestinal tract, thus reinoculate himself; the dosage thus administered is subliminal and insufficient to produce a generalized anaphylactic reaction, but can result (as Auer has shown) in a localized anaphylactic reaction at the point of irritation produced by the constant discharge of pus into the intestinal canal.

The acceptance of this theoretical assumption then should emphasize the need for a thorough investigation of any possibility of foreign protein sensitization in cases of obscure gastric hemorrhage or for foci of infection, especially those in organs emptying into the gastrointestinal tract, such as gall-bladder, appendix, or intestinal diverticulum.

SUMMARY

1. Definite hemorrhages from the gastrointestinal tract have been noted by many authors in which the point of bleeding could not be demonstrated.

2. Explanation of the hemorrhage in these cases merely on the basis of seepage from engorged blood vessels has never seemed entirely satisfactory.

3. Experimental work of Auer has suggested the possibility of the hemorrhage being a manifestation of auto-reinoculation of an individual previously sensitized by infection of the gall-bladder, appendix or diverticulum of the intestinal tract, with a resulting localized anaphylactic reaction.

4. All patients having hemorrhages from the gastrointestinal tract without demonstrable organic lesion should be subjected to thorough examination for foreign protein sensitization and for focal infection of organs which drain into the intestinal tract.

REFERENCES

1. AUER, J. Local autoinoculation of the sensitized organism with foreign protein as a cause of abnormal reaction. *J. Exper. M.*, 1920, xxxii, 427.
2. AUER, J. On the causation of experimental, anaphylactic hemorrhages in the stomach. *J. Pharmacol. & Exp. Therap.*, 1922, xix, 255–256.
3. BEVAN, A. D. Haemorrhage from the stomach. *Surg., Gynec. & Obst.*, 1924, xxxviii, 358–359.
4. CRISPIN, E. L. Toxic gastric hemorrhage. *Calif. State J. M.*, 1917, xv, 308–312.
5. DALCHÉ. Quoted by Monod (Ref. 12).
6. DEAVER, J. B. Gastric haemorrhage. *Surg., Gynec. & Obst.*, 1914, xiii, 294–299.
7. DRYBROUGH-SMITH, E. Note on hematemesis as a complication of appendicectomy. *Lancet*, 1918, ii, 15–16.
8. EUSTERMAN, G. B. The essential factors in the diagnosis of chronic gastric and duodenal ulcers. *J. Am. M. Ass.*, 1915, lxv, 1500–1503.
9. FINZI, O. Ueber Veränderungen der Magenschleimhaut bei Tieren nach Nebennierenextirpation und über experimentell erzeugte Magengeschwüre. *Virchow's Arch. f. path. Anat. (etc.)*, 1913, ccxiv, 413–432.
10. FRIEDENWALD, J., and WIEST, P. F. Massive hemorrhage from the stomach produced by unusual cause. *Ann. Clin. Med.*, 1924, iii, 158–161.
11. LATOUR. Quoted by Monod (Ref. 12).
12. MONOD, GUSTAVE. Haematemeses "without lesions." *Brit. M. J.*, 1924, i, 658–660.
13. MOSCHCOWITZ, A. V. Massive hemorrhages from the stomach without demonstrable ulcer. *Am. J. M. Sc.*, 1916, cli, 714–727.
14. NICOLAYSEN, K. Irritation of the vagus and hemorrhagic erosions of the stomach. *Arch. Int. Med.*, 1920, xxv, 295.
15. OUTLAND, J. H., and CLENDENING, L. Hematemesis due to chronic appendicitis, with an explanation of its pathological physiology. *Am. J. M. Sc.*, 1917, cliii, 273–275.
16. ROBINEAU. Quoted by Monod (Ref. 12).
17. ROSENOW, E. C. Elective localization of streptococci. *J. Am. M. Ass.*, 1915, lxv, 1687–1691.
18. SMITHIES, F., and BOWEN, R. The clinical significance of vicarious gastrorrhagia. *Illinois M. J.*, 1916, xxx, 126–132.
19. WHITE, W. H. Gastrostaxis, or oozing of blood from the mucous membrane of the stomach. *Lancet*, 1906, ii, 1189–1195.

VOL. XXI, No. 2

BONE LESIONS IN SARCOID: A ROENTGEN AND CLINICAL STUDY*

BY HOWARD P. DOUB, M.D., AND FRANK R. MENAGH, M.D.

Departments of Roentgenology and Dermatology, Henry Ford Hospital

DETROIT, MICHIGAN

THE lesion to be described in this communication is a form of tuberculosis sometimes affecting the osseous system. The etiology is frequently unrecognized, partially because the lesion is uncommon and partially because it is difficult to establish the etiological factor by the ordinary laboratory methods. This is a systemic lesion but it frequently has an associated skin manifestation, and descriptions of it are usually found in the dermatological literature. The bony lesions may be present, however, without involvement of the skin and therefore many cases of this type are probably missed, as the bony lesions are often relatively benign.

We present no new findings of primary importance in this disease, and none have been reported in the recent literature. We intend rather to draw attention to the roentgen changes in the osseous system as these have been emphasized but little in the roentgen literature. A brief description of the associated findings will be given.

CASE 1. G. D. (No. 72326). This patient, a male, aged sixty, was admitted complaining of swelling of the fingers. He was of American stock, formerly in the retail grocery business but had retired because of inability to use the hands. The family history was unimportant. The patient had always been fairly healthy until the onset of this present illness. This was eight years previous to admission when he developed a "fungus growth" on the mucous membrane of the nose. This caused nasal obstruction and was removed surgically. Shortly after this he noticed a swelling on the middle phalanx of the right index finger. Other bones were involved but only in the hands and feet. There was a moderate degree of reddening, pain and tenderness. The nails on the fingers and toes were rough and irregular and there was at times some exudation of serum. Concurrent with these changes lesions appeared in the skin of the face. They were deep in the skin, a purplish red, painless and had never suppurated. They had persisted practically unchanged for several years.

On physical examination the patient was an emaciated white man looking fully his stated age. Weight 133, thirty pounds under weight; temperature, 97°F.; pulse, 88; respirations, 20.

He seemed weak and ill. There was a marked stridor of the voice and on exertion there was marked dyspnea. The eyes were normal in function and innervation. Hearing normal. Upon perforation of the septum, there was found a brownish black growth incrusting both sides of the septum. The sinuses were clear on transillumination. The tonsils showed nothing unusual. The mucous membrane was pale but clear. A few small glands were palpable in the cervical and axillary regions. The thyroid was not enlarged. The chest was very thin and emphysematous but clear. The heart was not enlarged and no murmurs were heard. There was some thickening of the arteries. Blood pressure, 120/75. An electrocardiographic tracing showed some slight left ventricular preponderance. The abdomen was normal. The liver and spleen could not be felt. Examination of the rectum and genitalia revealed nothing unusual. The deep reflexes were active and equal.

The skin presented three distinct types of lesions:

(1) An area, 1.5 × 3.5 cm. over the right temple. This had been present since birth, and recently a small ulcer developed following an injury. This area was composed of closely grouped flat-topped papillae, soft and of a dark red color. In the center of the larger area, there was a small superficial ulcer, 0.8 cm. in diameter with a tight scab. The edges were raised, slightly rolled and indurated. This lesion comprised a typical non-vascular nevus with a superimposed basal cell epithelioma. The latter disappeared after treatment with radium.

(2) On the face there were several lesions which had persisted for at least six years, although there was no assurance that their morphology had remained constant. There was one lesion occupying the tip of the nose, one on either cheek, and two smaller lesions, the one on the chin being removed for section. The largest of these lesions, the one on the left cheek, was 4 cm. in diameter, while the smallest

* Read at the Twenty-ninth Annual Meeting, AMERICAN ROENTGEN RAY SOCIETY, Kansas City, Mo., Sept. 25–28, 1928.

was 1.5 cm. The lesions were dark purple in color and were slightly raised above the surface of the surrounding skin. They were smooth and showed no nodules. On inspection they appeared to have regular and well-defined borders. On palpation they were slightly indurated and without sharp edges. Over these lesions there was a fine network of dilated capillaries. The lesions were painless and showed no elevation of temperature.

FIG. 1. Case 1. Bone lesions in sarcoid. This case shows both bone destruction and trabecular alteration.

(3) The third type of lesion was found on the fingers and toes. On the right hand, the first, second, and fourth fingers were involved, while on the left the first and second fingers showed changes. The involved fingers showed fusiform swellings. The locations of the swellings varied on the different fingers but on one or another finger proximal, distal and mesial phalanges were all involved. On the right hand, the distal phalanx was involved in both the second and fourth fingers. Here the nails were atrophic and were exuding serum. The involvement of the second finger on the left hand was most marked. Here there was definite bone destruction. These lesions on the fingers were red, the skin tense and function was very materially interfered with. There was not much pain except on deep pressure.

FIG. 2. Case 1. Bone changes in phalanges of toes.

Both great toes were involved. The lesions were of the same type but not so extensive as in the fingers and there was no loss of function.

FIG. 3. Case 1. Fusiform swelling of fingers with involvement of nails of two fingers.

The laboratory reports showed the Wassermann reaction negative with all antigens. Blood count showed Hb. 69 per cent; red blood cells, 3,460,000; white blood cells, 5,300. The differential count showed: polymorphonuclears, 73 per cent; eosinophiles, 3 per cent; small mononuclears, 2 per cent; large mononuclears, 21 per cent; transitionals, 1 per cent. Blood chemistry showed: sugar, 59; nonprotein nitrogen, 31.6; urea N. 15.4; uric acid 2.90 mm. per 100 c.c. Urinalysis: specific gravity 1.002, reaction acid, albumin negative, Benedict negative, casts none. Microscopic examination was negative except for a few epithelial cells and leucocytes. The urine was negative for Bence-Jones proteins. These examinations have been frequently repeated without material change in the results noted. Smears made from the nose showed no acid-fast bacilli. An examination of the spinal fluid showed it to be normal. Tuberculin tests were negative.

Roentgen examination was made of both hands and both feet. There are multiple bone changes in the phalanges of the right thumb, index, middle and small fingers of the right hand. The right third metacarpal is also involved and there is involvement of the phalanges of the index and middle fingers of the left hand. The bones of the right foot also show involvement. These changes are characterized by a general alteration in the trabecular structure and by circumscribed areas of central destruction. There is also absorption involving the distal phalanges of the right middle and small fingers and the middle phalanx of the middle fingers of the left hand.

Roentgen examination was made of the skull, chest, spine and other long bones.

The skull is negative.

The chest shows only a moderate amount of perivascular fibrosis. The findings are otherwise negative.

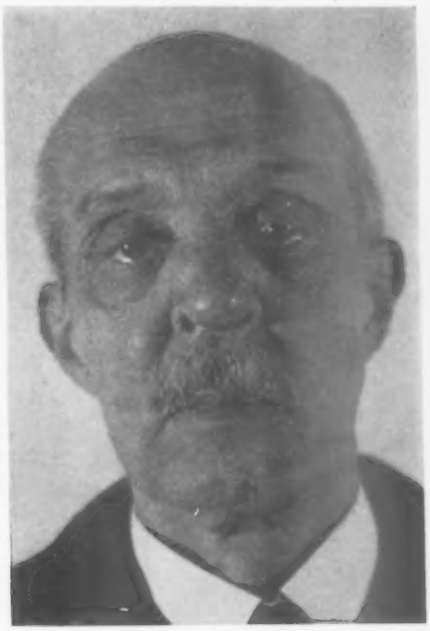


Fig. 4. Case 1. Shows patient when first seen early in 1926.

The pelvis and spine are negative.

A few cystic areas are seen in the lower end of the radius and ulna. No other pathology is seen.

A biopsy was done, a section of the skin being removed from the face for microscopic study.

Gross Pathology. The specimen consists of a small piece of facial skin 20 × 11 mm. in size. In the center an area 6 × 6 mm. is somewhat thickened without altering the surface characteristics of the skin. On cut through center no macroscopic changes are seen.

Microscopic. Sections show the surface epithelium of usual thickness but with some papillary hypertrophy throughout. Immediately about the papilla there is considerable round and wandering cell infiltration. Deeper we see rather large collections of round and wandering cells mingled with polymorphonuclear leucocytes. Still deeper we see single and conglomerate whorls of epithelioid cells with occasional giant cells forming rather typical tubercles. These lesions extend down to the line of excision. There is no caseation made out. The left second finger was also removed for study.

Gross Pathology. The specimen consists of left middle finger. It is enlarged to twice the normal size. The skin is unbroken but edematous. On section the skin and other soft tissues are pale, edematous and very soft. There is no exudate visible but a peculiar granular appear-

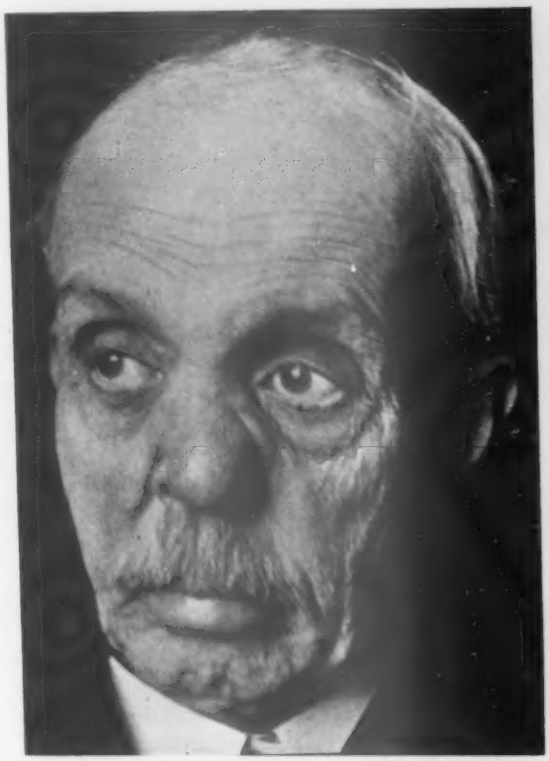


Fig. 5. Case 1. Patient two years later showing deformity of nose due to absorption of the cartilage.

ance is evident. On cut through specimen after hardening the bone is of pinkish-gray color, differing but little from color of soft tissues.

Microscopic. Sections taken from finger through skin, subcutaneous tissues and bone show the surface epithelium much thickened and covered with pink staining, homogeneous, epithelial debris. Deep in the areolar tissue we see single and conglomerate whorls of epithelioid cells with an occasional giant cell giving the typical appearance of tuberculosis without caseation. In the bone proper the marrow is entirely replaced by single and conglomerate whorls of epithelioid cells. Here also we see only an occasional giant cell and no suggestion of caseation. Sections stained for acid-fast bacilli fail to reveal the same on careful and systematic search.

Bacteriological. A cubic centimeter of tissue from the center of the bony portion of the finger was ground up in a mortar with salt

solution. This material was equally divided and injected directly into the inguinal glands of four half grown guinea pigs. These pigs continued to gain in weight and showed no glandular enlargement for a period of three months.

The skin lesions were first seen in January, 1926. Since that time the lesions have slowly subsided. The lesions on the face show much less coloring and there is not so much induration. The fingers are less swollen. The nails are still atrophic. The function in the fingers has not returned.

With the disappearance of the swelling in the fingers the skin has become tight and atrophic, the fingers a dead white and there is marked impairment of the circulation in the fingers. There is no flushing of the fingers in the dependent positions and they are not painful. They are sensitive to cold.

During this period of observation another fact has been noted. Without ulceration the cartilage of the nose has gradually disappeared. The stridor of the voice, noted on admission, has increased. Laryngoscopic examination shows a curious deformity of the larynx due to absorption of the cartilage here.

The patient himself during this period of observation has become extremely emaciated but shows no signs of active tuberculosis.

During this period various methods of treatment have been tried. The patient was given neo-arsphenamine intravenously. The hands were given roentgen therapy. Arsenic was given by mouth as Fowler's solution and Asiatic pill.

The patient's general emaciation and weakness was given consideration, but there is no evidence that any of these measures were of great value. As noted, the skin manifestations were somewhat improved but on the whole the patient has lost ground.

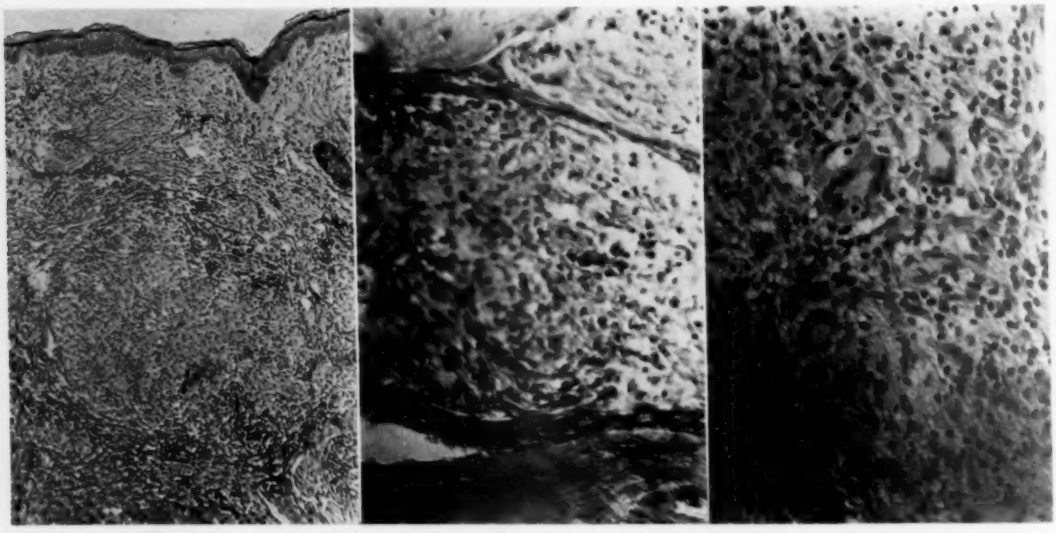


FIG. 6. FIG. 7. FIG. 8.

FIG. 6. Case I. Section from skin. Intense infiltration of round and wandering cells throughout areolar tissue and a whorl of epithelioid cells.

FIG. 7. Case I. Section from phalanx showing trabeculae of compact bone and complete replacement of marrow by whorls of epithelioid cells.

FIG. 8. Case I. Whorls of epithelioid cells and typical giant cells.

FIG. 9. Case II. Shows punched out areas in the bones with some areas showing bone reaction.

CASE II. F. S. (No. 86225). The patient is a white male, aged thirty. Occupation, machinist. Entrance complaint was "fits," gastric distress and headaches. The family history was unimportant. The patient stated that

about six years before admission a lesion appeared on the right cheek. This lesion has never been itchy or painful. Two years ago a similar lesion appeared on the opposite side of the face. Painless swelling developed on the fingers during this period. These have never interfered with his work.

This patient proved to be a typical case of Froelich's syndrome with impaired mentality and judgment and a clouded sensorium. Examination of the head, chest, cardiovascular system and abdomen revealed nothing unusual. The nervous system was involved with evidence of pituitary and cerebellar damage with roentgen evidence of change in the sella turcica.

The laboratory studies showed nothing of importance except a decreased sugar tolerance. So far as could be observed there was no evidence of tuberculosis, and the finding in the skin and tissues of the fingers could not be connected with the Froelich syndrome present.

This patient is obese. The skin is well filled out with the fat pads characteristic of pituitary disturbance. The hair shows a female type of distribution. The skin is dry. There are two types of skin lesions noted:

(1) There is a lesion on the right cheek, 2 × 5 cm. in size and of six years' duration. The edges are irregular and the surface rough. The color is a faded purple. The lesion is soft, painless and shows no evidence of any acute inflammatory process. The irregular surface is due to depressions between small soft nodules. There is no evidence of ulceration or scarring.

There are remains of similar lesions on the nose and opposite cheek. These are smaller in size and have nearly disappeared.

(2) All of the fingers on both hands, except two, show painless, fusiform swellings of six years' duration. The lesions are firm, not hard, although there is involvement of the bone as well as of the soft tissue. There is some curving of the nails, a beginning atrophic change. The function of the fingers has not been disturbed.

This patient was followed only a short time, and there was no opportunity to treat him or follow the evolution of these lesions.

Roentgen examination of both hands disclosed rather typical lesions in a number of the phalanges. These bones show a general alteration in the trabeculae, and one or two of the bones disclose punched out areas of destruction without much evidence of bone reaction. There was moderate soft tissue swelling present.

The lesions in the skin in both of these cases were of the same type. They were indolent, painless, non-ulcerating granuloma, involving chiefly the subcutaneous tissue. These lesions are sarcoids of the Darier-Roussy subcutaneous type. There have been several excellent descriptions of these lesions in the recent literature by Finnerud[1] and Goeckerman.[2]

ROENTGEN CHANGES

The bone changes, as shown roentgenologically, consist of a slowly progressive alteration in the trabecular formation of the bone followed by actual bone destruction as the lesion progresses.

There would appear to be several types of bone involvement although these may blend into each other in the later stages. In the one type, the primary lesion appears to be mainly an alteration in the trabecular structure of the bones which show a coarser architecture than the normal. As the lesion progresses, areas of destruction appear in the bones. These are either central or cortical or both, and vary in size from areas a millimeter or more in diameter to complete destruction of the bone.

In the other type, the lesions begin as punched-out areas of bone destruction in the phalanges. These may extend entirely through the bone and there is occasionally a line of eburnation around the margin. It is entirely probable that these two types are different manifestations of the same process and have the same end-result.

The phalanges are most frequently involved, although the metacarpals and metatarsals also show lesions and one of our cases had a small area of involvement in the lower end of one radius.

Complete destruction is most likely to occur in the terminal phalanges although other phalanges may also show extensive destruction. Cases are reported in the literature which showed tuberculous foci in the lungs, but the lungs were clear in our cases.

The progress is very slow as shown by the slight changes which developed in one of our cases under observation over a period of two and one-half years. There is usually moderate soft tissue swelling over the affected areas.

HISTOLOGY

The histological picture is rather typical

of a productive type of tuberculosis without caseation. The most constant typical findings are the whorls of epithelioid cells with occasional giant cells. The nodule-like areas may be single or conglomerate and are usually separated by connective tissue septa. There may be thickening of the surface epithelium and beneath this are often found collections of round and wandering cells mixed with polymorphonuclear leucocytes. These findings, however, are usually incidental to the typical epithelioid whorls. In a well-developed case the findings are rather typical, but in early cases they may suggest only chronic inflammation. Tubercle bacilli can usually not be demonstrated.

ETIOLOGY

While the etiology is not indisputable, most of the evidence, both clinical and experimental, points to the tubercle bacillus as the etiological factor. The histological picture is definitely that of a productive type of tuberculosis. Kyrle[4] has demonstrated the tubercle bacillus in early skin lesions, but in most cases this is not possible, especially in the later stages of the disease. Schaumann,[5] who has done extensive experimental work in an attempt to establish the etiology, has been able by inoculating guinea pigs and following them through successive generations to produce typical tuberculous lesions. He believes that the bovine strain may be the type involved, which might explain the unusual reaction. From the fact that various observers report a filtrable form of the tubercle bacillus we are led to suggest that the etiology may be explained on this basis. Schaumann's work would tend to confirm this opinion. This should be an interesting subject for future investigation.

DISCUSSION

The lesion under consideration would appear to be an atypical form of tuberculosis which may or may not show skin manifestations. The etiology has been under dispute, but those claiming the tubercle bacillus as the etiological agent would appear to be in a favorable position as tubercle bacilli have occasionally been demonstrated in the lesions, and guinea pig inoculations will sometimes produce the lesions. The histological picture is also typical of tuberculosis. These positive findings would appear to bear more weight than the negative ones. The tuberculin reaction is commonly negative unless the patient develops clinical evidence of tuberculosis.

The roentgen changes are fairly characteristic and will frequently clinch the diagnosis in an otherwise doubtful skin lesion. The bone lesions may be present without any other manifestation of the disease. The pathological picture in well-developed cases is that of tuberculosis without caseation.

This clinical picture would appear to include a group of lesions such as the Boeck and Darier-Roussy sarcoid, lupus pernio, and others. It is already apparent that it is a serious question as to whether these names should not be abolished and that this syndrome should be called tuberculosis with skin and bone manifestations. Jüngling[3] has called this ostitis tuberculosa multiplex cystica which would seem to be a better name than the classification used at present except that it implies bone changes only.

REFERENCES

1. FINNERUD, C. W. A clinical, histologic and bacteriologic study of a case of multiple benign sarcoid of Boeck-Darier-Roussy. *Arch. Dermat. & Syph.*, 1921, iv, 342–352.
2. GOECKERMAN, W. H. Sarcoids and related lesions; report of 17 cases; review of recent literature. *Arch. Dermat. & Syph.*, 1928, xviii, 237–262.
3. JÜNGLING, O. Ostitis tuberculosa multiplex cystica (eine eigenartige Form der Knochentuberkulose). *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1919–21, xxvii, 375–383.
4. KYRLE, J. Die Anfangsstadien des Boeckschen Lupoids; Beitrag zur Frage der tuberkulösen Aetiologie dieser Dermatose. *Arch. f. Dermat. u. Syph.*, 1921, cxxxi, 33–79. Über die tuberkuloiden Gewebsstrukturen der Haut. *Arch. f. Dermat. u. Syph.*, 1920, cxxv, 481–536.
5. SCHAUMANN, J. Benign lymphogranuloma and its cutaneous manifestations. *Brit. J. Dermat.*, 1924, xxxvi, 515–544.

ABSTRACT OF DISCUSSION

DR. RICHARD H. SUTTON, Kansas City, Mo., stated that our knowledge of the sarcoid of Boeck is as yet ill defined. He stated that he had gradually arrived at the opinion of the majority of observers that the condition is tuberculous. There are many, however, who

are not yet convinced of the tuberculous nature of the disease. This is due to the fact that although giant cells are found and the histological picture in the typical Darier-Roussy type is that of tuberculosis, still in the majority of instances the disease cannot be produced in animals by inoculation. The typical sarcoids of Boeck were at one time thought to react specifically to arsenic but this has been found not to be the case. Some of them do not respond to any type of treatment.

Dr. H. J. Ullmann, Santa Barbara, Calif., stated that he had seen one case of a sarcoid of Boeck which reacted favorably to small doses of roentgen rays.

Dr. L. R. Sante, St. Louis, Mo., called attention to a rare type of primary periosteal tuberculosis associated with skin lesions. The disease is apparently multiple and the roentgen picture is usually negative since only the periosteum is involved. The skin lesions are in the vicinity of and appear to be a direct continuation of the periosteal involvement. They look like rounded nodular elevations varying usually in size from 0.5 cm. to 3 or 4 cm. in diameter, are bluish in color and have the appearance of a malignant lesion which has broken through the skin from pressure. He had recently seen such a case in which the lower end of the radius and several places on the ribs were involved, producing an extrapleural effusion which had the appearance in the roentgen picture of a secondary manifestation of malignancy in the pleura. To further carry out this deception there were several round nodular areas scattered throughout both lung fields that simulated in every respect the appearance of a secondary malignant process in the lung. On careful roentgenoscopic examination and carefully tracing out these areas it was found that they were all connected with ribs and were actually extrapleural. Tubercle bacilli were found in the secretions from the wounds.

SARCOMA OF THE UTERUS*

BY J. A. CORSCADEN, M.D., AND A. P. STOUT, M.D.

NEW YORK CITY

INTRODUCTION

THE object of this communication is to report three cases of sarcoma of the uterus, and to study the importance of this disease as an actual menace to life, especially when developing in fibromyoma of the uterus. Sarcoma in the ordinary sense is a malignant and fatal disease. If it occurs in fibromyoma of the uterus as frequently as is indicated in statistical reports of recent years, one of which places the incidence as high as 12 per cent, there is needed a radical change in our attitude toward the management of the benign condition. According to the prevailing opinion, a simple uncomplicated smooth muscle tumor of the uterus of small size and without symptoms is not considered a menace. It is handled with the same alert conservatism as any other benign growth such as a wart or mole and becomes an object of treatment only when it shows changes or give symptoms. If it is a fact that a growth which would eventually destroy the patient occurs in 2 or more per cent of myomata, then the incidence of this growth reaches a higher figure than the operative mortality figure of supravaginal hysterectomy. Such a situation would demand the prophylactic removal of all myomatous uteri until there is discovered some symptom or sign which could be relied upon to herald the sarcomatous change. Myoma occurs in about 40 per cent of women reaching the age of forty. Consequently to be consistent we should operate on nearly every other woman of that age for this reason alone. Also, the addition of roentgen rays and radium to the list of therapeutic measures employed in the treatment of uterine myomata adds to the problem. After radiotherapy in proper dosage, cessation of uterine bleeding and shrinkage of the myoma may be expected in nearly all cases. The method, where available, has replaced

* Read at the Thirteenth Annual Meeting, American Radium Society, Minneapolis, Minn., June 11–12, 1928.

operation in about half the cases needing treatment. With such a satisfactory and widely practiced method at our command, the question of the subsequent malignant change of the tissue left *in situ* is of immediate importance.

In approaching this problem the first and most important effort is to define sarcoma. We may then attempt to estimate its frequency according to the criteria settled upon and, lastly, collect those cases of sarcoma which have followed the use of radiotherapy for fibromyoma, believing that in the host of cases so treated and observed the incipient sarcomata which, according to the statistics, should be there, would develop into definitely malignant growths, if indeed they were potentially malignant in the first place, and that we might thereby arrive at the approximate number of myomata which have turned malignant.

DEFINITION OF TERMS

Incidence among Uteri Removed at Operation and Autopsy

A very great diversity of opinion apparently exists as to what the words "sarcoma of uterus" connote. Because of this confusion, differences of opinion have arisen as to the proper method to employ in the prophylaxis and treatment of this disease. By subjecting some of the figures and statements recorded in the rather voluminous literature of this subject to a critical analysis, it is believed that it will be possible to point out the sources of some of this confusion. At the same time, this analysis will, we hope, present our own conception of this process.

No attempt will be made to discuss the various hypotheses regarding the origin of the tumor cells in the different types of neoplasms, first because there are widely divergent views held by different observers, and secondly because we cannot see that a solution of this problem will materially affect our knowledge of the biologic aspects of the question. We have therefore retained the old term of sarcoma, using it to cover all of the malignant, non-epithelial neoplasms of the uterus which have been described under this and a variety of other names.

By "malignant" we mean a growth which infiltrates the tissue beyond its capsule, metastasizes to distant parts of the body, or recurs after removal. That is, we have not accepted as of proven malignancy any tumor which showed gross or microscopic evidence of changes within the confines of the tumor proper, alone, unless it recurred following removal either locally or as a metastasis. Our experience and a study of the reported experience of others leads us to believe that it is impossible at the present time to recognize malignancy in this type of neoplasm by means of the microscope alone.

When the science of pathology was expanded in the second half of the nineteenth century, following the development of the methods of staining cells and tissues for microscopic study, the existence of a form of malignant neoplasm was recognized, which grew in the uterus usually in association with a fibromyomatous tumor and which was capable of invading contiguous tissues and of metastasizing to distant organs. Morphological investigation revealed that the cells composing the majority of these neoplasms resembled the parent cells of the fibromyoma but that they were very prone to vary in size, shape and number of nuclei; that grossly the neoplastic tissue was apt to degenerate, and that such degeneration was often associated with hemorrhages or cyst formation.

The first studies of these cases were made from autopsy material, but with the development of the modern operation of hysterectomy, surgeons began to remove vast numbers of uteri with fibromyomatous tumors. A certain number of these neoplasms were examined and among them were some which showed apparently the same cell changes as had been seen in the metastasizing sarcomata studied at autopsy. These changes were considered sufficient evidence to warrant the classification of these tumors as sarcomata.

A very extensive résumé of this whole period was made by Piquand (1905). Between the years 1857 and 1905 he collected reports of 137 cases, including 5 of his own. Of these, the diagnosis was based upon *morphological evidence alone* in 103. Of the remainder, 4 must be dis-

carded because of doubt as to whether they are sarcomata or carcinomata; 8 were found to have invaded surrounding structures; 15 recurred after removal and in 8 there were metastases. Therefore in this forty-eight year period, a compilation covering the literature of eight centuries contains records of 31 *proved malignant* sarcomata of the uterus.

Four years later Kelly and Cullen (1909) published another elaborate investigation. Among over 1400 myomata of the uterus, there were 17 which they considered showed morphological evidence of being sarcomata. An analysis of their reports shows that there were *proofs* of malignancy in but 7; i.e., 2 recurred locally after hysterectomy, 3 showed apparent invasion of neighboring tissues and 2 had distant metastases. On the other hand, a few of the 10 cases for which there exists nothing but morphological evidence that they were sarcomata, have remained well several years after operation.

In 1920 the results at the Mayo Clinic were studied by Evans. He found that from 1906 to 1918 inclusive, a period of thirteen and a quarter years, there had been 4000 operations for uterine fibromyomata. Seventy-two of these had been reported as being suspicious or malignant but only 11 proved to be so by recurring locally and invading the peritoneal cavity, and, using his criterion, only 13 were histologically malignant. This gives us a figure of 0.39 per cent of all fibroids removed by operation which are malignant. This corresponds very closely with the figure of 0.5 per cent obtained for Kelly and Cullen's proved malignant neoplasms among the fibromyomata.

A few years ago it began to be the fashion for pathologists and gynecologists to examine series of fibromyomata removed at operation for evidence of "sarcomatous degeneration," and the results were startling. Warner (1917) and Miller (1913) found that about 2 per cent of all myomatous uteri show "sarcoma," and Geist (1914), while examining 250 myomata found that 4.8 per cent of them showed "sarcomatous change." Spencer in discussing a presentation of 2 cases of sarcoma of the uterus by Andrews (1921–22) stated that he had found sarcoma in 6 per cent of all cases of myoma and that Bumm put the figure as high as 12 per cent and that because of this he now did total hysterectomy for myoma. All of these reports and others like them are based upon morphological changes and in few, apparently, has an attempt been made to find out what happened to the patient after she left the hospital.

If the observations of these writers are correct, then a considerable number of women who have retained myomatous uteri are afflicted with the disease "sarcoma of the uterus." If this is a malignant neoplasm we should be able to find autopsy records of such cases. At Bellevue Hospital, New York City, there are none recorded among the autopsy protocols, while there are only 3 cases, reported in this paper, at the Presbyterian Hospital, New York. At the Boston City Hospital in 1917 there was recorded only one case, which was studied by Gardner. He noted that of 827 leiomyomata examined at the hospital, 24 were histologically malignant but this was the only one which invaded or metastasized. Jacquin (1921) has reported 4 cases which showed proof of malignancy; these were from the Strasbourg clinic covering a period from 1909 to 1921. It seems to us that these figures, together with the reports of Piquand, Evans, and Kelly and Cullen, are convincing evidence that relatively few women die because of this malignant neoplasm.

It may be argued that hysterectomy has cured many of these women who would otherwise have died. But if this is true, why have we such meager autopsy records of women dying of sarcoma of the uterus in the days before hysterectomy for fibromyomata became such a common practice?

One cannot escape the impression that although an appreciable number of uteri may be found in which histologic changes have occurred exactly similar to or closely resembling the changes found in proved malignant neoplasms arising in fibromyomata, only a small proportion of these are actually malignant themselves. One cannot say what the proportion of malignant neoplasms among histologic

sarcomata of the uterus is, because seemingly no one has seen enough cases and allowed them a sufficient length of time. Our impression is that the figures of Evans are very close to the truth and that only about two-fifths of 1 per cent of all fibromyomata become malignant neoplasms.

CRITERIA OF MALIGNANCY

Granting that there is this difference in the biologic behavior of these neoplasms, it becomes of the greatest importance to attempt to find criteria of malignancy. We shall consider here only those neoplasms arising in relation to fibromyomata because the other types of sarcomata of the uterus, such as the one that is said to arise from the stroma cells of the endometrium, are admittedly so rare as to exist only as a few isolated cases.

A study of the evidence on record seems to show that if there is invasion of the tissues or veins outside of the fibromyoma, the chances of recurrence or metastasis are very great. But if the tumor is confined within the fibromyoma—that is, if there are histologic changes alone—there seems to be no sure way of predicting whether or not the neoplasms will recur or metastasize.

An attempt was made by Proper and Simpson (1919) to differentiate their cases of sarcomata into three classes of malignancy according to the degree of variation of the cells from the morphology of the simple leiomyomata. But it is obvious that such a classification must vary with each individual observer. A much more ambitious attempt was made by Evans. By estimating the number of mitotic figures per cubic millimeter he found that all of his proved malignant cases fell into a group in which the mitoses varied from 2,200 to 12,000 per cu. mm. Two unproved cases also fell into this group. All the rest showed mitoses varying from 0 to 800. This method is of interest because it is an attempt to provide a biometric criterion which can be used by anyone. Unfortunately it fails as a criterion in our Case 11, and there are a number of reported cases in which it fails also.

Kelly and Cullen have shown in their case reports that the sarcomatous changes in a fibromyoma may be so slight as to be overlooked in the cursory examination to which most fibromyomata removed by operation are subjected and yet the neoplasm may recur in the vaginal vault two years after hysterectomy. But they have also shown that tumors which have a more malignant histologic aspect may give no signs of recurrence nine years after removal.

Further, we cannot feel sure that an apparently simple fibromyoma may not manifest malignant tendencies. Jacquin (1921) has reported a case of neoplasm of the uterus which was histologically a pure myoma and which, ten months after hysterectomy, had a recurrence extending from the vagina to the liver with death eleven months after operation. He also cites the cases of Langerhans, Minkowsky, Schlangenhaufer and v. Franque which were fibromyomata of the uterus with metastases of adult fibromyomatous tissue in the lungs and elsewhere. Recently Brewer (1921) has reported a case of a fibromyoma occurring in the abdominal scar nine years after hysterectomy for fibromyomata of the uterus. It was his opinion that there was implantation of tumor tissue in the abdominal wound at the time of the first operation.

RECAPITULATION

(1) There are malignant neoplasms which seem to originate from within fibromyomata which are sometimes, but not always, characterized by degenerations, hemorrhages, mitoses and changes in the size, shape, number of nuclei, and arrangement of the cells, and which show a marked tendency to invade the surrounding tissues and veins and sometimes to metastasize through the blood stream; (2) these are very rare neoplasms occurring probably in less than half of 1 per cent of the fibromyomata removed by operation; (3) from 1.5 to 11.5 per cent of all fibromyomata removed by operation show morphological changes more or less closely resembling these proved malignant neoplasms, but statistical studies show that they probably cause no more embarrassment to the host than do other fibromyomata; (4) there have been recorded a

few cases of histologically pure fibromyomata which have shown invasive growth and have metastasized; (5) unless there are evidences of invasive growth or metastasis, there seem to be no unfailing criteria of malignancy in neoplasms arising in fibromyomata; (6) the terms "sarcoma," "malignant myoma" and their synonyms, all of which terms suggest malignancy or potential malignancy, have been applied indiscriminately to all of the above-mentioned types of neoplasms and this has led some to express the opinion that a good many fibromyomata are malignant tumors.

For the above reasons and because these terms "sarcoma" and "malignant myoma" have the same significance as cancer in the minds of most clinicians, it is our belief that the opinion expressed by Strong (1916) is equally true today: "The only safe criterion for the diagnosis of sarcoma of the uterus is infiltrative and destructive growth. Mere richness in cells, mitoses and even irregularities in the size of cells do not constitute sarcoma."

THE INCIDENCE OF SARCOMA AMONG FIBROMYOMATA TREATED BY RADIOTHERAPY

The difficulties of deciding what would have been the future behavior of a piece of tissue by its microscopic appearance are obvious. The appearance of the tissue must be compared with that of specimens from tumors which have in fact become malignant. In sarcoma of the uterus the total number of specimens, even on the broadest calculation, is so small as to make generalizations unsatisfactory.

To determine the frequency with which fibromyomata change to sarcoma, a study of a large series of cases of fibromyoma with a view to finding out their subsequent fate would be interesting. We have such a series in those cases treated by radium and roentgen rays. Since 1900 radiotherapy has been employed for the treatment of fibromyoma. In 1909 it had reached such a stage of development that it had become universally employed in Europe, and since 1914 has been increasingly used in the United States. At a single meeting in May, 1922, from five clinics were reported over 3000 cases. As nearly as can be calculated over 40,000 reported cases have been treated by radiotherapy.

We have thus a large number of potential sarcomata, and according to the most conservative estimate would expect to find reported a fair number of cases, during twenty years, of patients suffering from sarcoma of the uterus who had previously had radiotherapy. As a matter of fact, the following are the only cases which we have been able to cull by a careful search of the literature.

Case A—Hinterstoisser (1920). A woman forty-one years of age, was treated by deep roentgen therapy. She was compelled by the Communistic disturbances in Austria to move to another town so that the details of the treatment are not known.

Three weeks after the roentgen treatment was ended she was seized by severe signs of inflammation in the lower abdomen, with tenderness, no fever. Physical examination showed a mass, the size of a child's head. Operation: complete hysterectomy, leaving in the adnexa.

Pathological examination showed an intramural, well-encapsulated tumor of the body of the uterus. On cut section it showed a light tissue with bloody and black streaks through it, softened through necrosis.

Histological Examination. The tumor tissue which was fairly sharply demarcated from the uterus wall, consisted partly of spindle cells, partly of polymorphous cells, occasionally giant cells. In different places the smooth muscle cells seemed to be changing directly into the spindle cell sarcoma. (Malignancy not proved.)

Case B—Jacquin (1921). A woman, aged forty-eight, suffered for three years with menorrhagia, urinary symptoms and anemia. She had a uterus the size of a four months' pregnancy. The uterus was treated with roentgen rays. The bleeding stopped but not the urinary symptoms.

Six months later she was operated upon, the uterus excised. She declined rapidly, and died with metastases. The tumor showed polymorphous sarcoma with giant cells developing in a previously existing fibromyoma.

Case C—Shoemaker (1915). A woman fifty-six years old, menstruated regularly, and suffered with a sudden increase in amount, also suppression of urine. A large mass was found filling the lower abdomen.

Electric treatments gave no relief. A series of roentgen treatments of small dosage reduced

the mass one-fourth its size and controlled the bleeding within a year, but the mass grew again and two years later had nearly reached its previous size.

It was again treated with roentgen rays and diminished in size. No symptoms for five years when there was a recurrence of dark brown vaginal discharge. Operation: Removed a multinodular uterus which showed many varieties of cell pictures and mitoses but no metastases or tissue growth outside of the capsule of the fibromyoma. It is not stated whether one or more of the myomata in the uterine wall showed the tissue change described.

CASE D—See our report of Case III.

The occurrence of such a small number of sarcomata in fibromyomata of the uterus which had been treated by roentgen rays and radium is explainable on one of two theories: (1) That their actual incidence is very low, much below that suggested by the specimens removed at operation, or (2) that in some way or other the use of roentgen rays and radium has terminated in the myomata such processes as might have eventually ended in a true sarcoma. The doses used for the treatment of fibromyomata are so small that they do not cause the permanent disappearance of sarcoma in other tissues. It is difficult to believe that these cells are so much more susceptible. It seems fairer to interpret the scarcity of the malignant sarcomata among the cases under discussion as indicating an actual rarity of the disease.

CONCLUSIONS

Malignant neoplasms arising from fibromyomata of the uterus and broad ligament are very rare, occurring in from 0.39 to 0.5 per cent of operated cases.

Many thousands of reported cases of fibromyoma of the uterus have been treated by radiotherapy in the past thirty years. During this period there have been reported 4 cases of sarcoma of the uterus in fibromyomata which have received radiotherapy.

There are no clinical criteria by which sarcoma of the uterus may be recognized nor are there any infallible pathological criteria other than infiltrative growth or metastasis. Because the terms "sarcoma of the uterus," "malignant myoma" and their synonyms suggest cancer, their dogmatic use should be restricted to proved malignant neoplasms.

Since the operative mortality of hysterectomy is as great as or greater than the incidence of malignant neoplasms arising from fibromyomata, this operation as a prophylactic against possible subsequent development of malignancy is not justifiable.

The menace of sarcoma should not alter the present policy of alert conservatism in the management of fibromyoma nor should it prevent the use of radium and roentgen rays for these tumors.

CASE REPORTS

CASE I. Malignant tumor apparently developing from a fibromyoma of the broad ligament.

M. M. (P.H. Nos. 21678 and 21812.) Aged forty-six.

History. Previous diseases: none. Menopause one year before onset of present illness. Five normal pregnancies.

Present History. One month gradual onset of difficulty in starting urination, passing only small amounts. One week complete stoppage, catheterization necessary. Constipation of long duration especially severe for one month. Pain in lower abdomen, tearing, severe, and usually associated with desire to urinate, and sense of fullness in bladder region. Slight anorexia one week. Other symptoms negative.

Physical Examination. Mass connected with uterus filling pelvis, about 15 cm. in diameter, hard, tender, nodular. Nodules on cervix.

Pathology at Operation by Dr. G. E. Brewer. Some free black blood in peritoneal cavity. Right tube, left tube and ovary and uterus were "normal." In region of right ovary was a mass 8 × 10 cm.; firm, friable, red, containing old blood clot; adherent to intestines. Two cysts 5 × 7 cm. in right broad ligament.

Procedure. Cysts and mass in right broad ligament excised together.

Cross Examination. The specimens are (1) the right tube with a portion of the right broad ligament. The tube is somewhat tortuous and the fimbriated extremity is closed. In the broad ligament are 2 cystic cavities with thin, smooth walls, containing clear straw-colored fluid and measuring together 11 cm. in diameter. Adjacent to the cysts and tube is a roughened reddened area with adherent blood clots from which the second specimen has apparently

been torn. (2) A mass 14 × 7.5 cm. At one end it is firm and smooth, while the other half has a rough, irregular surface revealing many cyst-like cavities. In some areas there is a yellowish mottling. Toward the end which is still covered with an intact capsule, the cut surface suggests an architecture of interlacing fibrous strands, as in a fibromyoma. With this piece there are 5 other amorphous masses of soft, friable, whitish tissue and coagulated blood, the largest of which is 8 cm. in diameter.

Microscopic Examination. Sections from the firmer end of the main mass show the characteristic arrangement of a fibromyomatous tumor, i.e., interlacing bands of smooth muscle cells and fibrous tissue in which the great majority are smooth muscle cells. Intermingled with these are cell forms of extremely bizarre morphology, mostly much larger than the smooth muscle cells, very irregular in shape, both of cell body and nucleus. The nuclear material varies greatly in its staining reactions, and there are a number of cells with several nuclei. In this region it is difficult to find any mitoses. As one approaches the softer areas of the mass these bizarre cell forms dominate the picture to a greater and greater extent until finally no orderly cell forms remain. Here there are evidences of degeneration in the form of pycnotic nuclei, vacuoles in the cell cytoplasm, disappearance of intercellular substance and infiltration with round cells. In this region an examination of 100 oil immersion fields shows an average of 1400 mitoses per cu. mm. (Evans' method). At a still greater distance from the recognizable fibromyomatous architecture there is complete necrosis; amorphous masses of pink staining material mixed with fibrin and red blood cells (Figs. 1 and 2).

Course uneventful, home on fourteenth day. Thirty days after operation began to vomit. Lost appetite; dysuria, pain, and difficulty with defecation returned. P.X. showed a dense diffuse nodular mass filling pelvis. She went rapidly down-hill and died three months after operation.

Summary. There seems to be good evidence that this neoplasm existed first as a fibromyoma of the right broad ligament. Changes seem to have occurred in its component cells, resulting in proliferative growth, first between adjacent smooth muscle bundles, extending peripherally through the confines of the capsule of the fibromyoma and broad ligament into the adjacent structures of the pelvis. The invasive growth has been accompanied by necrosis and hemorrhage.

Case II. A malignant tumor apparently arising from an intramural fibromyoma.

C. M. (P.H. No. 55623.) Aged fifty-two, married.

Previous History. Three normal children, two miscarriages. Menstruation regular up to the age of forty-three; uncomplicated menopause. Previous diseases negative except for colds. Patient is a strong healthy housewife.

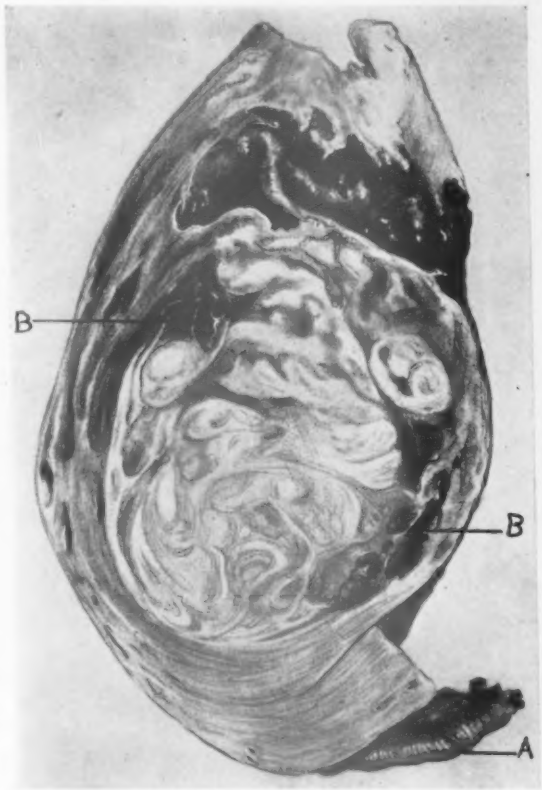


Fig. 1. Case I. Drawing of hemisection of uterus. The tube is shown at A. The large intramural fibromyomatous neoplasm is shown projecting forward from the posterior wall into the greatly stretched cavity. At B-B areas of necrosis with hemorrhage are shown.

Present History. Five months before admission to the hospital she began to have a little indigestion with discomfort after meals and belching of gas. For three months loss of weight (24 lbs.) and loss of strength. A month before admission a mass was felt by a physician in the lower abdomen but no significance was attached to it. For two weeks there had been a cough and sleeplessness. Four days before admission there was a little blood from the vagina and a tiny mass was felt. For four days there was a considerable dull pain in the lower abdomen. All of the symptoms were mild.

Physical Examination. A chronically ill

woman, coughs occasionally. Bad teeth, numerous skin moles, harsh vesicular breathing and subcrepitant râles at left base, a few moist râles over whole bronchial tree. Abdomen slightly distended, tympanitis, slightly tender in both lower quadrants. At the fourchette is a

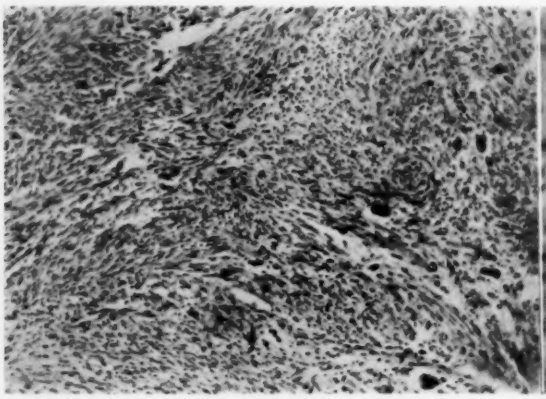


FIG. 2. Case 1. Photomicrograph of uterine tumor; interlacing bundles of smooth muscle and connective tissue. Scattered throughout can be seen cells of larger size with variations in the numbers and appearance of the nuclei.

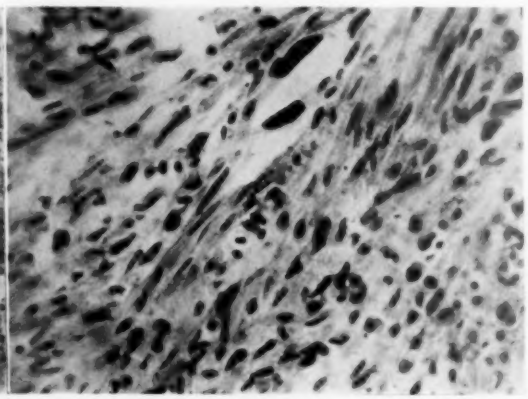


FIG. 3. Case 1. Higher power photomicrograph showing in more detail the variations in the size and appearance of the cells and their nuclei.

mass about 1 cm. in height, bluish color, soft, bleeds when touched. The uterus is 12 to 15 cm. in diameter, irregular, spheroid, firm, a little tender, fixed apparently by its size. Broad ligaments, and cul-de-sac clear. Temperature 102° F., pulse 90, respiration 22. White blood corpuscles 13,500; polymorphonuclears, 75 per cent.; red blood corpuscles, 4,100,000; hemoglobin, 60 per cent.

Diagnosis. Acute bronchitis; fibromyoma of uterus; papilloma of the skin of perineum.

Treatment. Advised to allow symptoms to subside before operating.

Course. Temperature rose irregularly to 103.4° F. on the fourth day after admission. Subjectively unchanged. Chest, unchanged. Blood culture, negative. Wassermann, negative. Stereoscopic roentgenograms show fields to be clear. Diaphragm irregular and costophrenic angle clear. There is no evidence of pulmonary disease.

Operation. Decided upon because sepsis was evidently increasing and coming from the uterus. The small pedunculated mass at vulva was excised. Dilatation and curettage before operation yielded some bluish greenish red material unfamiliar to operator. An immediate frozen section showed masses of small round cells. The probability of sarcoma was discarded because of the rarity of the condition. A massive inflammatory round cell infiltration in a myoma, and edema seemed to explain the picture. Laparotomy. Supravaginal hysterectomy, leaving in a thin rim of cervix and cutting the uterine vessels fairly close to the uterine wall. Double salpingo-oöphorectomy. There was an adhesion between the uterus and anterior abdominal wall. At this point was a reddish mass 2 cm. in diameter which looked like a firm red blood clot. The lower abdomen and broad ligament showed no infiltration or masses.

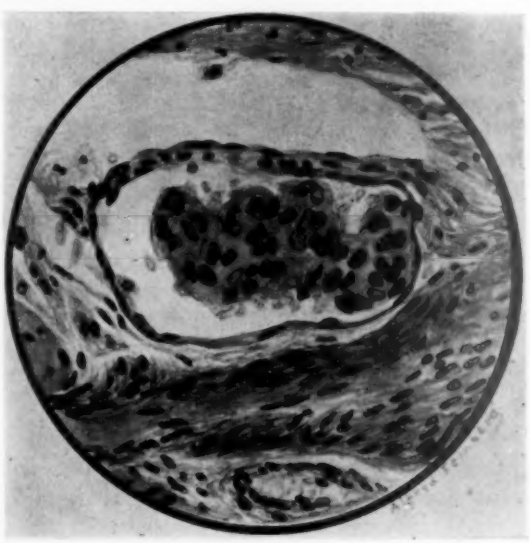


FIG. 4. Case 1. Drawing of vein from myometrium of uterus outside of the tumor showing plug of tumor cells attached to one wall and almost filling the lumen.

Gross Examination. The specimens are (1) a uterus removed without the cervix but with the tubes and ovaries attached. It is 10 cm. from fundus to cervix and 11 cm. broad. The

external surface shows the peritoneal surface intact over the fundus and upper part of the body, while the rest of the surface is covered with roughly torn tissue. The tubes and ovaries seem atrophic. An anteroposterior section from fundus to cervix shows that the increase in size

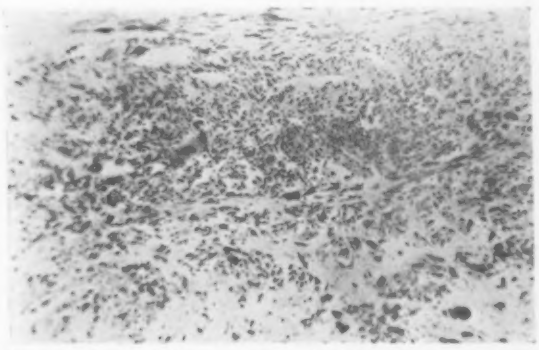


Fig. 5. Case 11. Photomicrograph of tumor tissue showing the interlacing bundles of smooth muscle and connective tissue. Scattered throughout can be seen cells of larger size and with irregularities in size and appearance of nuclei.

of the uterus is due chiefly to a large tumor 8 cm. in the longitudinal axis and 5 cm. anteroposteriorly. It lies in part between the inner layers of the stretched and thinned out muscle fibers of the uterine wall and impinges anteroinferiorly upon the uterine cavity. It has in general the appearance of a fibromyoma—that is, interlacing bands of fibrous and muscular tissue. Between some of these in the lower part, however, there are a number of dark reddish areas which seem to be made up of friable tissue and degenerated blood. Where the surface of the tumor in its lower part impinges upon the uterine cavity it is covered with ragged, shaggy remnants of degenerated tissue. The internal os is nearly 1 cm. in diameter and opens into the cavity of the uterus which is sickle-shaped, being displaced backward by the impinging tumor (see Fig. 3); (2) some friable blood clots said to have been removed from the area of adhesions between the anterior abdominal wall and the anterior wall of the uterus; (3) a mass of tissue 1 × 0.5 cm. said to have been removed from the perineum. It is dense and fibrous; (4) curettings from the uterus.

Microscopic Examination. Sections of the main tumor show approximately the same changes in all parts, the only variation being in degree. The usual architecture of the fibromyoma in which the smooth muscle cells predominate is preserved. Between adjacent bundles, however, there are groups of large cells with great variations in size and shape but with a tendency for the majority of the cells to be elongated. The cell bodies take a reddish brown stain as do the smooth muscle cells of the uterus and the fibromyoma. Most of the nuclei stain deeply and have an easily distinguishable reticular structure. Mitoses

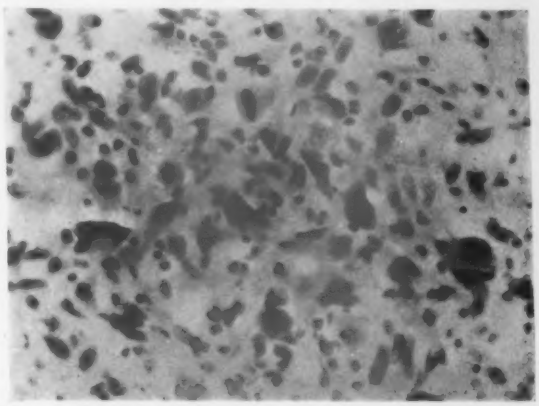


Fig. 6. Case 11. High power photomicrograph of tumor cells showing wide variation in the size, shape and appearance of the cells. There are three mitotic figures in the section.

vary greatly. In the area from which the photomicrograph was made, 100 oil immersion fields were examined without finding a single mitotic figure. In other parts of the tumor it is possible to find 3 to 4 in a single high power field. The general average everywhere is very low, however. The change from the uniform muscle cells of the fibromyoma to the adjacent cells of bizarre form is insensible; that is, the two types are intermingled at their junction (see Figs. 4 and 5).

A section from the wall of the uterus adjacent to the neoplasm shows some of the bizarre cells within the lumina of 3 separate venules. In each instance the cells are attached to the wall of the vein at one point but free elsewhere (Fig. 6).

(2) Sections of the tissue between the uterus and anterior abdominal wall shows blood clot which is filled in many places with cells of the same bizarre type. No mitoses found in this area.

(3) A section from the nodule in the perineum shows that the tumor has invaded this region also beneath a covering of thick stratified epithelium.

Course. There was a more or less continuous brownish discharge, with occasional gushes of blood. Induration in the broad ligaments increased. There was on the fiftieth day after operation, a definite globular friable mass in the stump. There was an abdominal fecal fistula on the tenth day after operation.

Bleeding from the vagina continued after operation. Signs in lungs were frequently heard, râles and rough breath sounds. No constant location. No signs of consolidation. Sputum occasionally blood-tinged. Course was progressively down-hill. Temperature remained elevated at all times. White blood corpuscles, 18,000 to 20,000. Pulse and temperature accorded with her general appearance. She had mild psychosis with delusions. She grew weaker and died on the sixty-ninth day after operation.

Summary. A history of gradual loss of flesh and strength in a woman, nine years after the menopause. Local symptoms of only a few days' duration. Bleeding apparently from a small lump in skin of perineum. The uterus felt as if it contained an ordinary tender myoma. Picture was not that of a metastatic sarcoma. At operation an acutely inflamed uterus was removed. There was an adhesion between it and the anterior abdominal wall, which, with the mass in skin of perineum, proved to be sarcoma. Death occurred sixty-nine days after operation and autopsy showed massive local recurrences as well as extensive lung metastases.

CASE III. A case of sarcoma in a uterus treated by radium and roentgen rays.

M. M. (P.H., No. 66078.) Aged forty-four, married.

History. Six months menorrhagia, irregular lower abdominal pain. Uterine mass 15 cm. in diameter. Hysterectomy advised, but because of an acute cold patient was given radium and roentgen treatment following diagnostic curettage, which was negative. Amenorrhea after one month. Pain in abdomen and slight temperature continued. Fourteen months after the first admission, profuse vaginal bleeding. Piece of tissue passed spontaneously. Operation: Diagnostic curettage, complete hysterectomy, double salpingo-oöphorectomy. Uterus nonadherent, contained a bluish mass of soft doughy consistence, about 8 cm. in diameter.

Pathological Report. Gross: The specimen consists of the uterus and cervix with left tube and ovary attached. Also the right tube and ovary and a portion of tissue removed from the interior of the uterus for frozen sections. The uterus measures 13 cm. by 8 × 6 cm. Attached to the posterior wall is a large tumor mass 7 cm. in diameter. The cervix and uterus have been opened through their whole extent and also the tumor mass. The uterine wall is thickened, firm and fibrous, 2 cm. in depth. A few Nabothian cysts are in the cervical canal. Projecting into the cavity of the uterus there is a cauliflower-like soft fibrous type of tumor growth, the lower surface of which is bluish black and necrotic. The upper part is a pale yellow, mottled. One portion of this tumor has evidently been removed from the lower extremity leaving a raw surface. At the point of tumor attachment to the lateral uterine wall there is a sharp line of demarcation between the tumor growth and uterine wall. The tumor mass on the posterior portion of the uterus is soft, lobulated and on section its capsule retracts. Many of the little lobules project as small rounded eminences and are pale yellowish gray. In between these nodules are excavated areas filled with soft cheesy necrotic material. This material appears to extend through the posterior wall into the uterine cavity. However, on section through the large tumor growth a portion of normal appearing uterine wall intervenes between it and the endometrial tumor in the uterine cavity. The tissue removed for frozen section is a soft, dark brown mass of what appears to be degenerated blood clot about the size of a small lemon. On one side of this mass is an excavation which is lined by a rough gray friable material. The left ovary is small, nodular and quite firm; the right ovary is small, firm and nodular and has on one surface a thick-walled cyst which on section exudes some dark brown jelly-like substance.

Microscopic: Section from the tumor in the interior of the uterus shows that it is composed of a mass of atypical tumor cells. For the most part these are somewhat fusiform in shape, but they vary enormously in size, shape and staining quality. Many of the cells have extremely large and irregular nuclei. A few multinucleated giant cells are seen. The tumor cells are growing in an irregular fashion, lacking any definite stroma. On one side of the section there is considerable necrosis and degenerated blood clot. Sections from the tumor on the posterior wall of the uterus show that it is composed of atypical tumor cells of a character similar to that found in the endometrial tumor. In some areas the cells are smaller, more fusiform in shape and have a more regular arrangement resembling the strands in a fibromyoma. In other areas the tumor cells are growing at random with large irregular nuclei and numerous mitotic figures. Mitotic figures average about 1 to 2 per high power field. There are numerous patchy areas of necrosis in this part of the tumor. Sections through the uterine wall at the junction of the tumor on posterior wall and tumor in endometrium show that the tumor cells from both

regions are penetrating the uterine wall along the fascial spaces between the muscle bundles. The sections at the junction of muscle wall and the tumor in the fibroid show some dilated capillaries partially filled with tumor cells. The large irregular tumor cells are not found toward the center of the uterine wall, but there are a few collections of pale blue staining fusiform cells scattered in irregular groups between the muscle bundles.

Diagnosis: Sarcoma of uterus.

Course. Uneventful convalescence. After nine months, hemoptysis. Roentgenogram shows metastases in the lung. Mass in pelvis appeared; increased in size. Patient died fourteen months following operation, with generalized metastases.

Comment. It is believed that this patient had sarcoma of the uterus in the first instance. Symptoms of pain and fever which continued after roentgen-ray and radium therapy were similar to those existing before any treatment was given.

BIBLIOGRAPHY

1. Andrews, H. R. Two cases of sarcoma of the uterus, with specimens and microscopical sections. *Proc. Roy. Soc. Med.*, 1910–21, xiv, *Sect. Obst. & Gynaec.*, 324–326.
2. Aubert, and Casse, E. Sarcome et grossesse. *Marseille-méd.*, 1919, lvi, 1126–1128.
3. Augier, D. Sarcome utérin à cellules polymorphes et à cellules géantes. *J. d. sc. méd. de Lille*, 1905, ii, 467–471.
4. Augier, D. Sarcomes de l'utérus et tumeurs à tissus multiples. *Gynécologie*, 1912, xvi, 213–219.
5. Bäcker, J., and Minich, K. Ein Fall von Sarkoma polyposum uteri. *Beitr. z. Geburtsh. u. Gynaek.*, 1909, xiv, 508–515.
6. Basso, G. L. Histologische Untersuchungen an einigen Fällen von Uterussarkom mit besonderer Berücksichtigung des Myosarkoms. *Monatschr. f. Geburtsh. u. Gynäk.*, 1907, xxv, 365–389.
7. Béclère, A. La radiothérapie des sarcomes de l'utérus à la clinique gynécologique d'Erlangen. *J. de radiol. et d'électrol.*, 1920, iv, 529–536.
8. Begouin, P. Sept cas de sarcome de l'utérus. *Rev. mens. de gynéc., d'obstét. et de pediat.*, 1912, vii, 371–373.
9. Berreitter, A. Zur Frage der Häufigkeit maligner Uterusmyome. *Zentralbl. f. Gynäk.*, 1921, xlv, 1592–1600.
10. Bettinger, L. Contribution à l'étude des sarcomes du col de l'utérus. Paris, 1909.
11. Bianchetti, F. Sarcoma a grappolo del collo uterina. *Minerva med.*, 1923, iii, 69–76.
12. Blacker, G. A case of supravaginal amputation of uterus for sarcoma mistaken for myoma. *Proc. Roy. Soc. Med.*, 1916–17, x, 111.
13. Brady, L. A sarcoma of the uterus arising from the endometrium. *Johns Hopkins Hosp. Bull.*, 1918, xxix, 164.
14. Brewer, G. E. Typical fibromyoma of the abdominal wall following hysterectomy. *Ann. Surg.*, 1921, lxxiv, 364.
15. Bureau, E. Sarcome de l'utérus. *Gaz. méd. de Nantes*, 1907, 2.s., xxv, 556.
16. Calderini, G. Un caso di sarcoma del corpo dell'utero. *Ann. di ostet. Milano*, 1916, i, 505–528.
17. Cauchoix. Sarcome fuso-cellulaire de l'utérus. *Bull. et mém. Soc. anat. de Par.*, 1905, lxxx, 478.
18. Chase, W. B. Sarcoma of the uterus. *Long Island M. J.*, 1911, v, 260.
19. Chatillon, F. Torsion axiale de l'uterus sarcomateux. *Schweiz. med. Wchnschr.*, 1920, l, 823.
20. Claessen, M., and Mathias, E. Beiträge zur Lehre vom Carcinosarkom. *Beitr. z. klin. Chir.*, 1921, cxxiii, 584–599. (Abs. in *J. Am. M. Ass.*, 1921, lxxvii, 2097.)
21. Clark, J. G., and Keene, F. E. Uterine hemorrhage of benign origin treated by irradiation. *J. Am. M. Ass.*, 1922, lxxix, 546–551.
22. Condon, A. P. Giant cell sarcoma of the uterine body. *Kansas City Index-Lancet*, 1908, xxix, 49–54.
23. Cullen, E. K. Combined adenosarcoma and mixed-celled sarcoma of the ovary. *Johns Hopkins Hosp. Bull.*, 1911, xxii, 367–369.
24. Cuzzi, A. Sarcoma del collo uterino trattata colla curieterapia. *Atti d. Soc. lomb. di sc. med. e biol.*, 1922, xi, 54–70.
25. Dahlet, E. Maligne Degeneration der Uterusmyome. *Ztschr. f. Krebsforsch.*, 1919–20, xvii, 536–555.
26. Dalton, H. C. Sarcoma of the uterus; report of a case. *Weekly Bull. St. Louis M. Soc.*, 1910, vi, 208.
27. Davies, T. B. Sarcoma of the cervix. *Proc. Roy. Soc. Med.*, 1914–15, viii, *Sect. Obst. & Gynaec.*, 43–45.
28. Dorland, W. A. N. Perithelial sarcoma of the uterus. *J. Am. M. Ass.*, 1908, li, 1227–1229.
29. Drew, D. Sarcoma of the uterus in a patient aged twenty-five. *Proc. Roy. Soc. Med.*, 1910–11, iv, *Sect. Obst. & Gynaec.*, 111.
30. Driessen, L. F. Fibrosarcoma uteri (with sarcomatous metastases). *Nederl. Tijdschr. v. Geneesk. Amst.*, 1919, ii, 1587.
31. Dubergey, J. Sarcome de l'uterus. *Gaz. bebd. d. sc. med. de Bordeaux*, 1920, xli, 331–333.
32. Erck, T. A. A case of sarcomatous degeneration of a fibroid tumor of the uterus with repeated hemorrhages into the tumor. *Am. J. Obst.*, 1905, lii, 889–892.
33. Evans, N. Malignant myomata and related tumors of the uterus; report of 72 cases occurring in a series of 4,000 operations for uterine fibromyomata. *Surg., Gynec. & Obst.*, 1920, xxx, 225.
34. Fabricius, J. Sarkom des Uterus. *Wien. klin. Wchnschr.*, 1917, xxx, 1117.
35. Froeschmann, E. Sarcoma adenomatosum uteri. *Ztschr. f. Geburtsh. u. Gynäk.*, 1919, lxxxi, 623–640.
36. Fukada, S. A case of a big sarcoma growing in round ligament. *Sei-i-Kwai M. J., Tokyo*, 1912, xxxi, No. 7, July 10.
37. Fullerton, W. D. Uterine sarcoma. *Surg., Gynec. & Obst.*, 1914, xix, 711–718.
38. Garcia de Arias, R. Un caso de fibrosarcoma del utero. *Rev. Ibero-Am. de cien med., Madrid*, 1910, xxiii, 27–33.
39. Gardner, L. U. A case of metastatic leiomyosarcoma primary in the uterus. *J. Med. Research*, 1917, xxxvi, 19–29.

40. GEIST, S. H. The clinical significance of sarcomatous change in uterine fibromyoma. *Am. J. Obst.*, 1914, lxix, 766–778.
41. GEIST, S. H. A contribution to the histogenesis of sarcomatous changes in uterine fibromyomata. *Am. J. Obst.*, 1913, lxviii, 1053–1063.
42. GRAD, H. Sarcoma of the uterus, with remarks on radiotherapy. *Am. J. Obst.*, 1905, li, 599–615.
43. GRIFFITH, W. S. A., and WILLIAMSON, H. A case of fibromyoma of the uterus undergoing sarcomatous change. *J. Obst. & Gynaec. Brit. Emp.*, 1906, ix, 84–94.
44. HEDLEY, J. P. A uterus containing a carcinoma and a sarcoma. *Proc. Roy. Soc. Med.*, 1916–17, x, *Sect. Obst. & Gynaec.*, 81.
45. HELLIER, J. B. A case of grape-like sarcoma of the cervix uteri. *J. Obst. & Gynaec. Brit. Emp.*, 1914, xxvi, 108–110.
46. HERRENSCHMIDT, A., and LEO, G. Un nouveau cas de sarcome kystique de l'utérus. *Bull. et mém. Soc. anat. de Par.*, 1907, lxxxii, 457–465.
47. HINTERSTOISSER, H. Nekrose eines Myosarkoms des Uterus nach Röntgenbehandlung. *Zentralbl. f. Gynäk.*, 1920, xliv, 977.
48. HOEVELS, K. Ein Fall von myoblastischem Sarkom des Uterus mit Lungen- und Nierenmetastasen. *Frankfurt. Ztschr. f. Path.*, 1911, viii, 477–498.
49. HOLLAND, E. A case of sarcoma of the uterus. *Proc. Roy. Soc. Med.*, 1908–09, ii, *Sect. Obst. & Gynaec.*, 344–349; *J. Obst. & Gynaec. Brit. Emp.*, 1911, xx, 56–59.
50. HORROCKS, P. Fibro-myoma of the uterus undergoing sarcomatous degeneration. *Tr. Obst. Soc. London*, 1905, xlvi, 184–188.
51. JACQUIN, P. A propos du sarcome et myome malin de l'utérus. *Gynec. et obst.*, 1921, iii, 90–111.
52. JEANNE. Sarcome kystique de l'utérus. *Bull. et mém. Soc. de chir. de Par.*, 1909, n.s., xxxv, 1004–1007.
53. JETTET, H. Sarcoma of the body of the uterus from a girl aged twenty. *Proc. Roy. Soc. Med.*, 1910, iii, *Obst. & Gynaec. Sect.*, 186.
54. JONES, A. T. Simultaneous occurrence of adenocarcinoma and sarcoma in the same uterus. *Am. J. Obst.*, 1911, lxiii, 449–459.
55. KEIFFER, H. Sarcome utérin. *Rev. mens. de gynec., d'obst. et de pediat.*, 1908, iii, 344–346.
56. KEIFFER. Fibromes multiples calcifiés et sarcome de l'utérus. *Gynécologie*, 1909, xiii, 135–138.
57. KELLY, HOWARD A., and CULLEN, THOMAS S. Myomata of the Uterus. W. B. Saunders Co., Philadelphia, 1909.
58. KLEE, F. Ein Karzinomsarkom des Uterus. *Zentralbl. f. Gynäk.*, 1922, xlvi, 166–170.
59. LATTEUX, and PROISST. Sarcome spacéle de l'utérus. *Bull. et mém. Soc. anat. de Par.*, 1909, lxxxiv, 305–307.
60. LECENE, P. Sarcome développé sur un utérus fibromateux; hystérectomie supravaginale; guérison, opératoire. *Ann. de gynéc. et d'obst.*, 1907, 2.s., iv, 663.
61. LECENE, P., and MONDOR, L. La myectomie large dans certains sarcomes primitifs musculaires. *J. de chir.*, 1911, vi, 616–626.
62. LOCKYER, C. *Proc. Roy. Soc. Med.*, 1907–08, i, *Obst. & Gynaec. Sect.*, 245.
63. MACDONALD, E. *Tr. Soc. Obst. & Dis. Women*, 1909, 83.
64. MAJOR, R. H. Multiple primary malignant tumors with a report of a case of carcinoma and sarcoma in the same individual. *Johns Hopkins Hosp. Bull.*, 1918, xxix, 223–231.
65. MARONEY, W. J. Sarcomatous change in uterine fibroids. *Am. J. Obst.*, 1916, lxxiv, 445–450.
66. MASOTTI, P. Sarcoma a cellule polymorfe della mucosa dell'utero. *Riv. veneta di sc. med.*, 1911, lvi, 337–346.
67. MASSON, J. C. Sarcoma of the uterus. *Am. J. Obst. & Gynec.*, 1923, v, 345–357.
68. MCFARLAND, J. Sarcoma of the vagina; a statistical study of 102 cases with the report of a new case of the grape-like sarcoma of the vagina of an infant. *Am. J. M. Sc.*, 1911, cxli, 570–588.
69. MERIEL. Sarcome primitif du corps utérin. *Toulouse méd.*, 1905, 2.s., vii, 253–260.
70. MEYER, R. Beiträge zur Pathologie des Uterussarkoms mit Demonstrationen am Epidiaskop. *Ztschr. f. Geburtsh. u. Gynäk.*, 1907, lx, 354–368.
71. MEYER, R. Ueber den Stand der Frage der Adenomyositis und Adenomyome im allgemeinen und insbesondere über Adenomyositis seroepithelialis und Adenomyometritis sarcomatosa. *Zentralbl. f. Gynäk.*, 1919, xliii, 745–750.
72. MILLER, J. R. The relation between sarcoma and myoma of the uterus and its bearing on x-ray therapy of uterine myomata. *Surg., Gynec. & Obst.*, 1913, xvi, 315–321.
73. MILLS, H. W. Sarcomatous degeneration of fibroid of the uterus. *Interstate M. J.*, 1918, xxv, 230.
74. MININGHAM, W. D. Sarcoma of the uterus in a child; operation; recovery. *J. Med. Soc. N. Jersey*, 1909–10, vi, 556.
75. MOENCH, G. L. Sarcoma of the round ligament. *Med. Rec.*, 1920, xcvii, 652.
76. MONOD, E. Sarcome de l'uterus. *J. de med. de Bordeaux*, 1907, xxxvii, 107.
77. MORRIS, R. S., and WOOLLEY, P. G. Sarcoma of the uterus. *Lancet-Clinic*, 1916, cxvi, 164.
78. M'WEENEY, E. J., and GIBSON, M. J. Sarcoma and endothelioma in the same uterus. *J. Path. & Bacteriol.*, 1907–08, xii, 132–134.
79. NAGY, T. Ueber ein Sarkom der Gebärmutter entstanden auf Grund einer infektiösen Granulombildung. Zugleich ein Beitrag zur Frage der Psoriasis und Leukoplakia uteri. *Arch. f. Gynaek.*, 1914, cii, 611–638.
80. NEEF, F.-E. Concurring tumors in women. *Am. J. Surg.*, 1916, xxx, 344–355.
81. NISOT, P. Sarcome de l'utérus et grossesse. *Gynéc. et obstét.*, 1922, v, 88–90.
82. NOBLE, C. P. Fibroid tumors of the uterus. *J. Am. M. Ass.*, 1906, xlvii, 1881.
83. NUTHALL, A. W. Sarcoma of the uterus. *Birmingh. M. Rev.*, 1912, lxxi, 184–186.
84. OUTERBRIDGE, G. W. The simultaneous occurrence of carcinoma and sarcoma in the uterus. *Am. J. Obst.*, 1917, lxxv, 575–591.
85. PAVESIO, C. Sarcoma dell'utero. *Riforma med.*, 1912, xxviii, 791–793.
86. PERDRIGE, J. G. Sarcomes de l'uterus. Lille, 1911.
87. PIQUAND, G. Des degenerescences des fibromyomes. Thèse de Paris, 1905.
88. PIQUAND, G. Le sarcome de l'utérus. *Rev. de gynéc. et de chir. abd.*, 1905, ix, 387–446.
89. POOL, W. P. Uterus containing sarcomatous degeneration of a fibroid and an independent adenosarcoma. *Am. J. Obst.*, 1916, lxxiv, 493.
90. PORTER, M. F. Sarcoma of the uterus, rectum, and adjacent tissues. *Fort Wayne M. J.*, 1906, xxii, 146.
91. PROPER, M. S., and SIMPSON, B. T. Malignant leiomyomata. *Surg., Gynec. & Obst.*, 1919, xxix, 39–44.
92. PROUST, and CARAVEN. Sarcome (myome-malin) de l'utérus. *Bull. et mém. Soc. anat. de Paris*, 1909, lxxxiv, 307–309.
93. RAABE, H. Zur Frage der Implantationsmeta-

stasen in der Laparotomienarbe nach Exstirpation karzinomatöser Genitalorgane, zugleich ein Fall von primärem Tubenkarzinom mit Metastasen in der Bauchdecken. *Beitr. z. Geburtsh. u. Gynaek.*, 1910, xv, 242–277.

94. Reel, P. J., and Charlton, P. H. Sarcoma of the uterus. *Ann. Surg.*, 1923, lxxvii, 476–484.
95. Robin. Thèse de Bordeaux, 1912.
96. Savariaud, Jacob, and Poulain. Sarcome dégénéré de l'utérus avec dégénérescence colloïd et calcification partielle. *Bull. et mém. Soc. anat. de Par.*, 1920, xc, 356.
97. Schirmer, F. A. Ueber die Verbreitung des Uteruscarcinoms im Kanton Thurgau und seine Behandlung im Kantonsspital Münsterlingen, Zurich, 1919.
98. Schmincke, A. Zur Kasuistik primärer Multiplizität maligner Tumoren. *Virchow's Arcb. f. path. Anat. (etc.)*, 1906, clxxxiii, 160–163.
99. Schottlander, J. E., and Kermauner, F. Zur Kenntnis das Uteruskarcinoms. Monographische Studie über Morphologie, Entwicklung, Wachstum, nebst Betriegen zur Klinik der Erkrankung, No. 43. Berlin, 1912.
100. Schultze, B. S. Die Axendrehung (Cervixtorsion) des myomatösen uterus. *Samml. klin. Vortr.*, 1906, Gyn. No. 152, 505–554.
101. Schumann, E. A. Observations upon the sarcomatous metaplasia of certain uterine fibromata. *Am. J. Obst.*, 1900, lix, 408.
102. Sicard, P. Sarcome primitif de l'utérus avec annexite double simulant une tuberculose caséeuse des trompes. *Bull. et mém. Soc. anat. de Par.*, 1908, lxxxiii, 453.
103. Shoemaker, G. E. Sarcomatous degeneration of a fibromyoma of the uterus 5 years after *x*-ray treatment for pressure and hemorrhage. *Tr. Coll. Phys. Phila.*, 1915, xxxvii, 142. Also *Boston M. & S. J.*, 1915, clxxiii, 210; *J. Am. M. Ass.*, 1915, lxiv, 1653.
104. Shoemaker, G. E. Sarcoma in the central portion of a uterine fibroma; syphilis. *Am. J. Obst.*, 1917, lxxv, 1046.
105. Smyly, Sir W. A case of myoma undergoing sarcomatous degeneration. *Med. Press & Circ.*, 1912, n.s. xciii, 85.
106. Solomons, B. A. Sarcoma of the uterus. *Tr. Roy. Acad. M., Ireland*, 1915, xxxiii, 327.
107. Spanton, W. D. Case of large myoma, with sarcomatous outgrowth; abdominal hysterectomy. *Med. Press & Circ.*, 1907, n.s. lxxxiii, 337.
108. Spencer, H. R. Carcinoma-sarcoma uteri. *Proc. Roy. Soc. Med.*, London, 1916–17, x, 82.
109. Spencer, H. R. Two cases of supravaginal amputation of the uterus for supposed myoma found to be sarcoma. *Proc. Roy. Soc. Med.*, 1916–17, x, 112.
110. Strong, W. L. Quoted by Maroney, W. J. *Am. J. Obst.*, 1916, lxxiv, 445.
111. Swayne, W. C. Uterine fibroids showing sarcomatous degeneration. *Proc. Roy. Soc. Med.*, 1907–08, i, *Obst. & Gynaec. Sect.*, 151–153.
112. Taylor, H. C. Sarcoma of the uterus. *Am. J. Obst.*, 1909, lix, 287.
113. Taylor, H. C. *Am. J. Obst.*, 1908, lvii, 116–118.
114. Taylor, H. C. Six cases of sarcoma of the uterus. *Am. J. Obst.*, 1907, lvi, 373–376.
115. Taylor, J. R. Uterine fibroid with sarcomatous degeneration. *Long Island M. J.*, 1911, v, 161–163.
116. Thaler. Sarkom und Karzinom im Uterus. *Wien. klin. Wchnschr.*, 1915, xxviii, 746.
117. Tracy, S. E. Fibromyomata uteri. *Surg., Gynec. & Obst.*, 1908, vi, 246–254.
118. Vanelli, V. Sarcomi dell'utero e loro istogenesi. *Gior. internaz. d. sc. med.*, 1915, n.s., xxxvii, 433–445.
119. Veit, J. Atiologie, Symptomatologie, Diagnostik, Prognose der Myome. Handbuch der Gynäk., J. F. Bergmann, Wiesbaden, 1907–08, ii, 487.
120. Verdier, Pierre. De l'hematometrie dans le cancer du corps uterins en particulier dans le cancer epithelial et le sarcome diffus dela muqueuse uterine. Paris Thesis, 1906.
121. Vincent and Laffont. Un cas de cancer à évolution rapide ayant succédé à deux séances de rayons X en fibrome utérin. *Bull. Soc. d'obst. et gynec. de Par.*, 1922, xi, 440–442.
122. Viques. Progres de méd., 1921, xxxvi, 289.
123. Virchow. Die krankhaften Geschwülsten. iii, 289.
124. Vogt, M. E. Sarcoma of the uterus with a report of thirty cases. *Am. J. Obst. & Gynec.*, 1923, v, 523–529.
125. von Kubinyi, P. Sarkomatöse Entartungen von mit Corpuskarzinom vergesellschafteten Uterusfibromen. *Pest. med.-cbir. presse*, 1911, xlvii, 39.
126. Warnekros. Vier bis fünfjährige Heilung fortgeschrittener Karzinome durch Bestrahlung. *Ztscbr. f. Geburtsh. u. Gynäk.*, 1919, lxxxi, 571–583.
127. Warner, F. Uterine fibroids with reference to their tendency to become sarcomatous; a study of 100 consecutive cases. *Am. J. Obst.*, 1917, lxxvi, 888–894.
128. Winter, G. Die malignen und benignen Degenerationen der Uterusmyome. *Ztscbr. f. Geburtsh. u. Gynäk.*, 1906, lvii, 8–66.
129. Witter, F. C. Sarcomatous degeneration of uterine fibroids with a report of eighteen cases. *J. Michigan M. Soc.*, 1916, xv, 565–572.
130. Young, J. V. D. Fibropolyp of the uterus undergoing sarcomatous degeneration. *Am. J. Obst.*, 1914, lxix, 1050.

TREATMENT OF CARCINOMA OF THE RECTUM*

BY T. E. JONES, M.D.

Cleveland Clinic

CLEVELAND, OHIO

THE end-results in a series of 65 cases of cancer of the rectum in which the treatment was by radium and roentgen rays are here presented. On account of the technical difficulties involved in the treatment of this condition and the apparent hopelessness of the outlook, this field has not been given as much attention as the more accessible regions, the skin, the oral cavity and the cervix. A discussion of this subject may therefore be of some interest.

The group included in this report consists of a series of 53 inoperable cases and 12 operable cases of cancer of the rectum in which the patients refused the radical operation. In every case treatment was given more than two years ago; more recent cases have not been included in this series. Time will not be taken here to discuss what constitutes operability; suffice it to say that the only cases referred for radiation treatment generally fall in one of the above-mentioned groups.

The technical difficulties which have been alluded to are frequently due to the inaccessibility of the growth or our inability to place the radium accurately and to hold it in place with complete protection of the uninvolved rectal mucosa. We are further handicapped by the fact that we are treating an organ whose physiological function is necessary while the reaction is going on. To ameliorate these symptoms, chiefly pain and tenesmus, colostomy has been advised, but the patient often objects to this procedure just as he objects to the radical operation. However, he can generally be persuaded to have the colostomy performed on the ground that it can be closed, should he be cured.

An analysis of the cases in this series shows that the youngest patient was twenty-three, and the oldest eighty years of age. There were 12 patients between the ages of forty and fifty, 25 between fifty and sixty, and 15 between sixty and seventy, the average age being fifty-four years. Fifty-six of the patients were married and 9 were single. There were 42 men and 23 women.

The average duration of the symptoms was 11.4 months. This, of course, is most regrettable and in most cases the delay in treatment was due to the fact that a digital examination of the rectum was not made by the examining physician. Many of these patients had sought medical advice because of bleeding and had been inadequately examined.

As stated above 12 of these cases were operable but the patients refused operation. Of these, 5 are living from three to seven years after treatment and 7 are dead. The average duration of life among those who died was twenty months, which is considerably longer than the average duration in untreated cases.

Fifty-three cases were inoperable; 5 of the patients are living and 48 are dead. The average duration of life in these 48 patients was fourteen months.

Colostomy was done in 57 cases. It should be advocated in all cases because it makes the patient so much more comfortable. Some of the patients who at first refused a colostomy submitted to it later when pain and tenesmus followed the treatment. The advantages of colostomy are that straining at stool is eliminated and the distal segment can be kept clean by through-and-through irrigation and all irritation avoided. In cases in which the growth is near the sphincter I have been able to give considerable relief from pain and spasm by performing a simple myotomy on the external sphincter, in much the same way as one would perform a subcutaneous tenotomy. A small incision is made about an inch from the anus and the scissors inserted down to the sphincter, which is divided. This procedure has the advantage of not leaving a raw surface at the mucocutaneous border.

In 2 out of the 10 cases in which a cure

* Read at the Twenty-ninth Annual Meeting, AMERICAN ROENTGEN RAY SOCIETY, Kansas City, Mo., Sept. 25–28, 1928.

has been effected I have been able to close the colostomy. One of these patients has been well for six years and the other for five years. In the other cases, the colostomy cannot be closed because the rectum has become greatly narrowed following treatment and I have not advised an attempt at dilatation because the strictures are very dense.

Treatment by radium has been carried out by three methods—tubes, needles, and seed implantation, and in some cases by a combination of all these methods. Time will not permit a detailed description of the method of application, because it must vary with the nature and location of the growth in each individual case.

As for the general treatment, the patient is hospitalized, and if there is no obstruction the bowels are cleaned out with frequent small doses of a saline laxative. If there is no gross metastasis in the liver or glands, a colostomy is performed, the sigmoid flexure being brought out through a muscle-splitting incision on the left side. This is opened two days later and immediately the distal loop is irrigated with large amounts of clear warm water twice a day. In about a week the radium treatment is given. If tubes or needles are used, I would call attention to two very important points: The treatment should be given under sacral anesthesia in order to get proper placement of the radium, and it should be done with the patient in bed so that he does not have to be moved after the radium has been properly placed. In this particular group of cases the larger the amount of radium applied, the easier it is for the patient. I think that the introduction of gold seeds will supplant the use of tubes and needles in the treatment of this condition on account of the more homogeneous radiation obtained and the ease of application.

The dosage varied in our cases from 1000 to 5000 mg-hr., the average being 2500 mg-hr. The number of treatments varied from one to five. In most of the cases the total dosage was given in two treatments. Screening was generally 1.5 mm. of brass in the tubes and 0.5 mm. of steel in the needles. The tubes and needles were placed tandem or parallel, according to the nature of the lesion.

Roentgen rays were used in addition to the radium in 45 of the 65 cases. Among the 10 patients who have remained well, 8 were treated by roentgen radiation and 2 were not. High voltage roentgen treatment was given by Dr. Portmann, and from one to ten treatments were administered, the amount being governed by the general condition of the patient.

Four of the deaths were due to intercurrent disease, 2 to apoplexy after nine months, and three and one-half years respectively, one to pneumonia after five months, and one to coronary thrombosis after two months. The other patients died of cancer, either as the result of extension into the pelvis or bladder or of metastasis to the liver or lungs. The average duration of life following treatment in all the cases in which death occurred was fifteen months.

In looking over these results it seems to me that much good has been accomplished and that the treatment is worth while. A follow-up study of these cases has shown that 7 of the men have recovered sufficiently to be able to resume work and have therefore been restored to economic independence, while the other one is comfortable at an age in which he is naturally unproductive. The two women are able to attend to all their household duties. Such results indicate that it is worth while. In addition to these results one must also consider the great relief that has been afforded for many months or years by palliation of the symptoms in the cases in which the patients died of the disease. Moreover, if we persevere, I think that greater good can be accomplished as our technique improves.

SUMMARY

1. Sixty-five cases of carcinoma of the rectum are reported in which treatment was by radium and roentgen radiation. In 10, or 15 per cent of the total number, the patients are well, having remained so for periods varying from three to seven years.

2. Although the treatment of choice for an operable cancer of the rectum is

operation by the combined abdominoperineal routes, if operation is refused or if the condition is inoperable, there is sufficient evidence that a cure can be obtained in certain cases and marked palliation in others by the use of radium and roentgen rays.

CANCER OF THE RECTUM

No. of Cases	Av. Age	M	F	M	S	Symptoms	Op.	Inop.
65	54	42	23	56	9	11 mo.	12	53

Colostomy	Radium	X-ray	Living	Dead	Average Duration of Life
57	65	45	10	55	15 mo.

CANCER OF THE RECTUM

Radiation

	Living	Dead	Average Duration of Life
Operable cases	12 5	7	20 mo.
Inoperable cases	53 5	48	14 mo.

Age Incidence

Decades	2d	3d	4th	5th	6th	7th	8th
Cases	3	6	12	25	15	3	1

Patients Living	Duration
1	7 yr.
3	6 yr.
3	5 yr.
2	4 yr.
1	3 yr.
Total 10	Average 5 yr.

MEASUREMENT OF RADIATION DOSAGE*

BY U. V. PORTMANN, M.D.

Cleveland Clinic

CLEVELAND, OHIO

AT the recent International Congress of Radiology held in Stockholm, a unit of roentgen-ray dosage, the "r-unit," was defined and adopted. It now becomes necessary for every therapist to employ some method for the measurement of dosage in the terms of this new international r-unit. This is not only essential as a scientific procedure but is also advisable from the medicolegal point of view. Dosimeters or other means for the estimation of dosage are not so generally employed in this country as they are in most European countries, where in almost every laboratory some kind of dosimeter is used either for constant measurement or for frequent periodic checks of the output of the roentgen apparatus.

Experience and experiments have taught us that dosage cannot be accurately expressed by means of the factors of voltage, amperage, filter and time alone, as they were formerly employed. There is a wide variation as to both quantity and quality in the output of different roentgen installations even when the same mechanical factors are used, and there may be also an occasional variation in the output of any given apparatus. The dosage of radiation therefore can no longer be expressed in the terms which have previously been employed, but to be accurate and to make satisfactory comparisons, the dosage must be stated in r-units, and the quality of the radiation must also be indicated. It would seem to be appropriate at this time for the American Roentgen Ray Society to insist on the use of this new method of expressing dosage in all the papers which are to be presented before the society though for a short time to permit the old factors to be included in order to prevent confusion.

The measurement of roentgen-ray dosage is no longer either a difficult or a complicated procedure. Periodically each roentgen therapy installation should be calibrated and standardized; the intensity output should be determined in r-units, and the quality should be defined for the various treatment techniques which are usually employed in that particular laboratory. Whenever any mechanical change in the installation is made, recalibration is necessary. If soft or intermediate qualities of radiation are used, this procedure is especially necessary when tubes are changed since there is great variation in

* Read at the Twenty-ninth Annual Meeting, AMERICAN ROENTGEN RAY SOCIETY, Kansas City, Mo., Sept. 25–28, 1928.

the output of different tubes under these conditions. Calibration of installations does not give an absolute determination of the dosage in r-units which is administered to each patient, not only because of the variations in the output of any apparatus but also because of the differences in those factors which influence backscattering, such as the size and shape of the field, and the volume of tissue which is irradiated. Periodic calibration, however, is much better than none at all and the approximate dosage may be estimated therefrom.

Unquestionably, the most satisfactory method of determining dosage is by means of an instrument which may be applied directly to the skin of the patient within the field of radiation; thus the radiation from the tube, as well as the backscattering, may be measured independently of the voltage, distance, amperage, filter, the size and the shape of the field, and the volume of the irradiated tissue. Such an instrument must be equipped with a small ionization chamber, and if different qualities of radiation are used, the chamber should be made of a material which will measure intensity, independently of the wave length employed; therefore, the chamber must have the same absorption for roentgen rays as air itself. This last requirement is suggested by Fricke and Glasser, who developed a chamber of the air-wall type. If only one quality of radiation is used with a particular apparatus, it is possible to employ an ionization chamber constructed of a material which has a greater absorption than air, but such a chamber should, of course, be calibrated and standardized in r-units for that quality of radiation employed and in terms of air absorption.

In small chambers, on account of the very limited amount of current produced by ionization, it is necessary either to use a very sensitive measuring device, such as an electrometer (electroscope), or to amplify the current so that a less sensitive galvanometer may be employed. This amplification of the ionization current is a complicated procedure and requires elaborate apparatus. In our experience any one of the various types of the easily operated string electrometers is efficient and rugged enough for practical clinical use. We believe that the roentgen dosimeter of choice is one which consists of a string electrometer with a static charging device and a properly insulated wire connecting the electroscope to an electrode which terminates within an ionization chamber of the air-wall (Fricke-Glasser) type. Such a dosimeter may be equipped with a chamber of such volume as will produce an ionization current so small that the total dose may be registered for a considerable period of time.

There is another type of dosimeter which may be employed when hard radiation is invariably used with a particular therapy apparatus, or to check the constancy of output. This dosimeter consists of a large ionization chamber with multiple-plate electrodes, and since the ionization in such a chamber is proportionately large, a sensitive galvanometer may be used to measure the current produced. When this apparatus is calibrated and standardized in r-units, it will indicate the dosage which is being given during a specified time, but it cannot be used for soft radiation. Since it is placed within the tube container or usually directly under the filters, it registers only the focal radiation, and due allowance must therefore be made for backscattering, a factor which varies considerably under different conditions. Probably an ideal arrangement would be to use a large chamber with a galvanometer to control the constancy of current during treatment and a small-chamber apparatus for the measurement of the dose which is being administered to the patient.

Every dosimeter must be standardized in terms of the international r-unit and periodic checks are required in order to be certain of the constancy of the instrument. This check can be satisfactorily made in the therapy laboratory by the use of a radioactive standard. The radioactive standard control may consist of any convenient radium preparation or of a special container of uranium oxide or other radioactive compound, but it is essential to use the same standard for each calibration. The procedure of checking a

dosimeter by means of the radioactive standard is simple, since it is only necessary to place the control preparation at a special place which is usually provided for it on the electrometer or ionization chamber, and then to read from the scale the rate of discharge. This should be done at the time when the instrument is standardized. If there is a very small variation from that ionization which was noted at the original standardization of the instrument, then proportionate allowance must be made in calculating the dosage. Considerable variation in the rate of ionization indicates the presence of some defect in the instrument, which should be corrected.

The original calibration and standardization of any dosimeter should be made by comparison with a standard large air-ionization chamber, or with another dosimeter which has been thus calibrated and is known to be constant and accurate. The large air-ionization chamber is purely a laboratory instrument, or a standardizing control, and it is complicated in its construction and operation. Since it is not a clinical apparatus, the therapist need not be familiar with the details of the method of its use. It is hoped that in the near future the Bureau of Standards at Washington and subsidiary stations throughout the country will be equipped with large standard air chambers and other apparatus such as is necessary for the calibration and standardization of dosimeters.

Although the international r-unit is at present only applicable to the dosage of roentgen rays, this unit may probably also be applied to the measurement of the radium dosages. Heretofore it has been practically impossible to compare the intensities of radium radiation with those of roentgen rays, because the instruments which are used to measure roentgen-ray intensities cannot be properly protected from the penetrating gamma rays. Thus electrometers are discharged by radium even at a distance and when they are heavily protected. Nor do the large standard air chambers give accurate comparisons with radium because of the impossibility of proper protection and the scattering effects produced within the air chamber itself.

In our research laboratory we have developed an instrument by which we believe it is possible to make a satisfactory comparison of the intensities of radium radiation and of roentgen rays and to standardize the dosage of each in terms of the same physical r-unit. This instrument, which we call the "condenser dosimeter" (Glasser, Portmann and Seitz), was first reported at the meeting of the American Roentgen Ray Society held in Montreal in 1927. The apparatus is divided into two sections, the first being a string electrometer with a static charger to which may be attached a removable second section consisting of a small condenser in connection with a small ionization chamber of the air-wall type. In operating the instrument, the two sections together are charged by the static charger to a known potential, which is read on the scale of the electrometer; then the section made up of the charged condenser and the ionization chamber is removed and is taken to a place at any distance where the ionization chamber may be exposed to radium or to roentgen rays for a specific time. Thus the condenser becomes discharged by the ionizing effect of the radiation within the ionization chamber. This section is then reattached to the electrometer and the amount of discharge of the condenser is read upon the scale. The apparatus is calibrated in r-units and the intensity of the measured radiation is determined on the basis of the duration of the exposure. The dosimeter may be calibrated so that the r-units per second of exposure of the chamber may be read directly on the scale. Calibration of the instrument is made by comparison with the large standard air chamber.

With this condenser dosimeter Glasser has found that the threshold erythema dose with radium, as determined by Failla and Quimby, is apparently about 1600 r-units. This same erythema dose with roentgen rays, when backscattering is included, would seem to approximate 900 r-units. This difference between the effect of roentgen rays and of radium on the skin is to be expected, because of the greater quantity of the shorter and more penetrating wave lengths of radium.

Although we now have a physical unit which is applicable for the measurement of radiation intensities of both radium and roentgen rays and are able to determine the quality of radiation in the terms of wave lengths by means of the spectrograph or by the half-value layer in aluminum or copper, we still lack a biological standard, such as, for example, that used to standardize antitoxins. The first consideration in the standardization of dosage has necessarily been the universal recognition of a physical unit which would make it possible to compare and to understand the biological reactions when accurately measured dosage is used.

Up to this time it has been customary to employ the reaction of the human skin as a biological standard and to compare the reaction of other tissues to the skin erythema. We have therefore usually stated that a given number of erythema doses would be required to destroy certain neoplasms, although we did not know the actual quantity or quality of the radiation which was employed. It is apparent that the skin reacts differently to the same measured dosage with different qualities of radiation if considered over a wide range of wave lengths, since we have proved that with the grenz rays, 250 r-units will produce erythema, while with the gamma rays of radium, 1600 r-units are required. As far as the skin is concerned, it is certain that the short wave lengths influence the subcutaneous tissues to a greater degree than do the rays which are absorbed more superficially and that the reaction of the subcutaneous tissues must directly or indirectly influence the skin reaction to a certain degree. It is a question also as to whether or not neoplastic cells and other tissues will react differently under the influence of different qualities of radiation. This can be proved only by comparing the biological reactions to accurately measured radiation intensities with various qualities of radiation in the terms of our international physical unit.

In determining the dosage, we have in the past used this variable reaction on the surface of the skin as an indication of a maximum of 100 per cent dose and we have also compared the intensities of these depth doses on the basis of this uncertain biological standard. We have thus expressed depth doses in percentages of the erythema. In the light of our present knowledge, it would seem that the intensity of radiation which is administered to a deep-seated area should be expressed directly in r-units rather than in the percentage of the superficial dose, which, after all, gives no intimation of the actual dose which may or may not give a desired effect upon a deep-seated lesion.

Any discussion as to the number of r-units which are required to produce an erythema is somewhat academic except from the standpoint of determining a maximum skin tolerance, since really the important matter is the measurement or calculation of the intensity which may affect an area that is under treatment. The therapist may therefore choose the skin dosage which he regards as safe. An example of this method of expressing the roentgen-ray dosage would be as follows: If one should choose 800 r-units as the safe maximum skin dose with a specified quality of radiation and if 50 per cent of this dose, or 400 r-units, reaches the area under treatment through three portals, then the actual dose would be 1200 r-units, with due consideration for the volume of tissue under treatment.

What has been stated in regard to the dosage of roentgen rays may also be said of radium. We have heretofore expressed radium dosage, both as to surface and depth doses, in the terms of milligram-hours and of skin erythema instead of in the actual number of physical units which are administered. Although the designing of isodose curves for roentgen rays on the basis of r-units is not a difficult matter, in the case of radium it is a complicated procedure, because of the peculiar varieties of filters and applicators. However, it is also possible to develop for radium standard isodose curves which will permit the estimation of dosage in r-units. We are now working on this problem.

The advantages of expressing both the depth dose and the surface dose of radium and roentgen rays in terms of r-units are obvious, not only from a physical point of view but also from the standpoint of

investigation of biological reaction. Various types of neoplastic tissues react differently according to the various quantities of radiation used, so that at present we may seldom make a prognosis on the basis of cell type alone. For the same reason we have been unable to predetermine the amount of radiation which should be administered to a particular lesion in order to obtain the desired effect. However, as a result of researches at the Crocker Institute, Dr. Francis Carter Wood has been able to state that each of their experimental tumors was completely destroyed by a certain measured dose of radiation, and that when less than this specific quantity of radiation was employed, there was proportionately less effect upon the cells. The investigators were able to make a curve for each tumor, which graphically illustrates this proportionate dosage and its effect. Some tumors required comparatively small doses while others required doses of enormous intensity for their complete destruction, but in each instance there was a certain intensity which invariably completely destroyed the tumor cells. Thus, in the case of these tumors the lethal dose could actually be predetermined in physical units.

Since we now probably have a means of measuring the intensities of both radium and roentgen rays in the terms of one physical unit, we may expect that in the future we shall be able to express in uniform international standard terms the total dosage of the radiation either of roentgen rays or of radium, or of a combination of both. After longer experience gained by comparison of results and by researches based upon a better physical foundation, we shall also expect to find that in dealing with a particular type or grade of neoplastic cell, we should employ a certain predeterminable dosage to produce a desired effect. With due consideration of the extent and type of the disease, the volume of the tissue, and the depth of the lesion, this measured dose may then be administered by a proper combination of our sources of radiation, and radiation therapy will then become much more efficient.*

* For discussion of this paper see page 176.

THE RELATION OF FRACTIONAL TO DEPTH DOSE*

BY H. J. ULLMANN, M.D.

Director, Department of Radiology and Cancer Research, Santa Barbara Cottage Hospital

SANTA BARBARA, CALIFORNIA

RADIATION effect loss becomes of greater importance as evidence accumulates that results are improved when so-called tissue saturation is reached and maintained over a certain period of time. Such rate of loss, therefore, has become an additional factor in determining a roentgen dose and it was while calculating dosage with this additional factor that it was noted that the order in which the portals of entry were used had, under certain conditions, a marked effect on the relative depth dose. A number of tables were then made to determine if possible, theoretically, not only the optimum order of ports but also the optimum fraction of dose to be given to each port at each application.

This paper presents the results of such calculations for a single group of conditions. Nothing is offered to show that the absolute value of any one of the factors used is correct but the principle involved will hold for any value.

To determine the rate of effect loss comparative curves were plotted from the published graphs of Pfahler[1,2] and Stenström.[3] It will be seen that there is a considerable difference, not only between the graphs of the two authors but between the graphs of the same author (Pfahler). (Fig. 1.) Pfahler's graph was developed from clinical observation while Stenström's was plotted mathematically from the results of experiments. As the loss used

* Read at the Twenty-ninth Annual Meeting, AMERICAN ROENTGEN RAY SOCIETY, Kansas City, Mo., Sept. 25–28, 1928.

by Pfahler in his work is close to 6 per cent per day for the first few days when the average of both his graphs are plotted and that of Stenström is 8 per cent, a loss of 7 per cent per day was used in making these calculations. It is believed that, for the present, such a rate will be safer for practical application than 8 per cent, for we have no experimental evidence as yet for the rate of loss in any tissue except skin. Factors used in making the chart and other calculations necessary to this paper are assumed to have the following values:

Loss of radiation effect for all tissues:

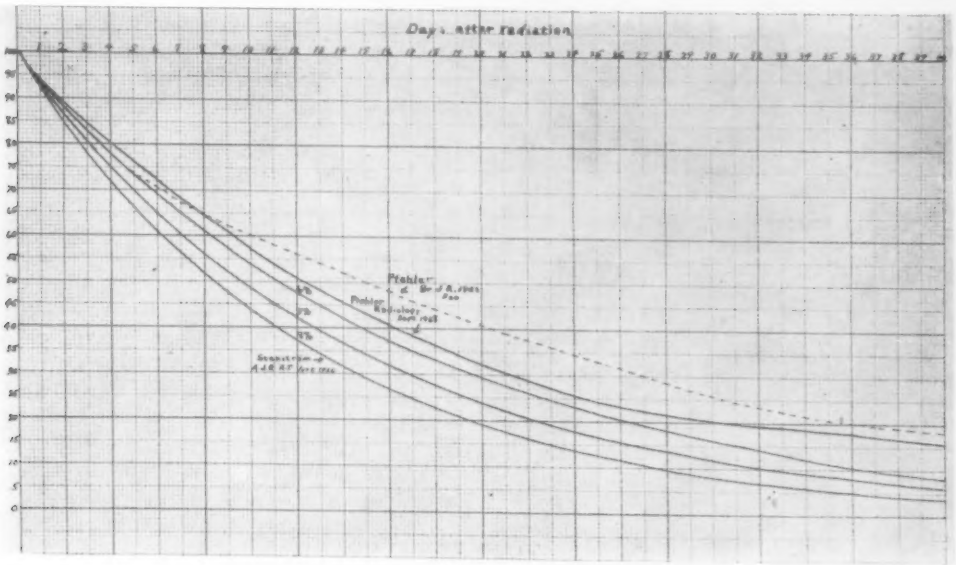


FIG. 1.

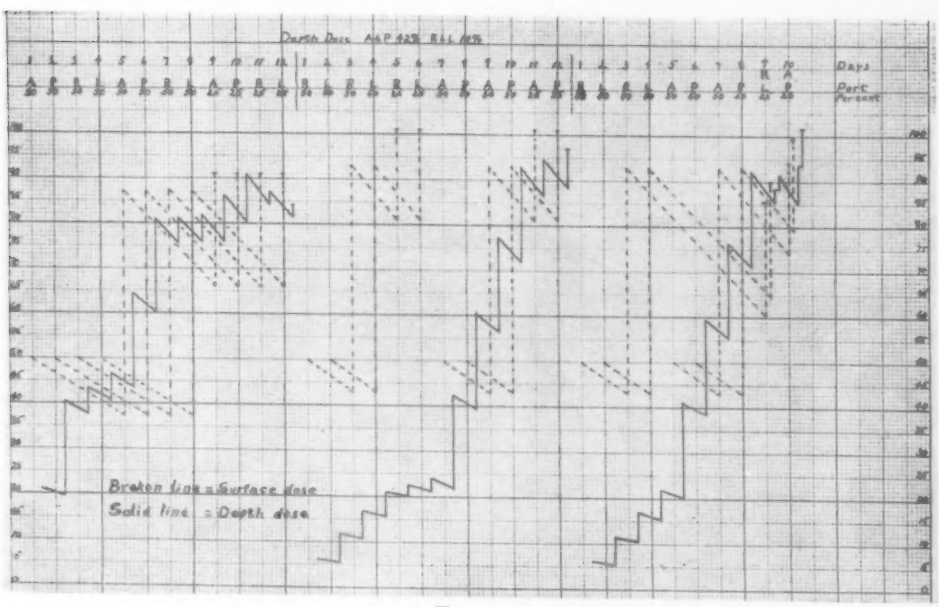


FIG. 2.

7 per cent per day. Measurements of body treated: anteroposterior 20 cm., lateral 36 cm. Region to be irradiated at center of body. Depth dose: 42 per cent of surface dose at 10 cm., and 12 per cent at 18 cm. No

allowance is made for the increase in surface dose due to irradiation through from opposite port.

Figure 2 shows the relation between depth and surface dose under three conditions where the order in which the ports are given is the only variant. The dose on the last day is decreased to prevent overdosage to the skin. In each graph it can be seen that the lateral port becomes less useful as the depth dose increases. This is obvious for the daily absolute loss must increase with increase of total quantity as treatment progresses while the amount given at each application remains constant. The last two lateral ports on the first chart are not only of no value but the depth dose actually falls as the skin dose increases. It is therefore better, on general principles, to use those ports first which give the smaller relative depth dose, and it is absolutely necessary to see that the fraction applied to any port is sufficient to give a depth dose greater than the loss at that depth since the last irradiation.

SUMMARY

The order in which portals of entry are used in crossfiring has marked effect on the total depth dose where there is considerable difference between the depth and surface dose and becomes of greater importance with increase of such difference.

CONCLUSION

The order in which the portals of entry are given should be specified when planning treatment of tissues at considerable depth. If this is not done the amount of radiation at the depth may be inadequate in spite of multiple ports and skin limit quantities.

REFERENCES

1. PFAHLER, G. E. Saturation method in roentgen-therapy as applied to deep-seated malignant disease. *Brit. J. Radiol.*, 1926, xxxi, 45–58.
2. PFAHLER, G. E., and WIDMANN, B. P. Further observations on the use of the saturation method of radiation therapy in deep-seated malignant disease. *Radiology*, September, 1928, xi, 181–190.
3. STENSTRÖM, W., and MATTICK, W. L. Study of skin reactions after divided roentgen-ray dosage. AM. J. ROENTGENOL. & RAD. THERAPY, 1926, xv, 513–519.

DISCUSSION OF PAPERS OF DR. PORTMANN AND DR. ULLMANN

DR. A. U. DESJARDINS, Rochester, Minn. I agree with Dr. Portmann that it is highly desirable that we should use some physical basis of recording dosage, but the chief value of this will consist in our added ability to understand one another.

In reading papers, for instance, it is very difficult to get more than a rough idea of what the other man is doing at the present time, whereas, with some such scheme as this, we may be able to come a little closer. However, that is only one phase of the problem, and there are several others.

The next great argument which will be filling the discussions at our societies will be the number of r-units for this and that, and there will also be—and I think I can predict this with a certain amount of confidence—a certain number of r-units for this tumor and that tumor.

I cannot have any great sympathy, or cannot fall in with this idea at all because not only can we talk of the type of cell but we must talk of the variations in the same kind of tumors, and the same kind of tumor varies a great deal. For instance, in carcinoma of the breast metastasizing in the spine in two patients approximately alike, in one patient you can cure the condition, and in the other you cannot do it. Why? Because of individual variations in the tumor in that patient. You can give as many r-units or as few r-units as you like, but you will have the variations taking place just the same.

So far as the present scheme of recording dosage is concerned, I admit the great desirability of using a physical basis, but when it comes to criticizing the actual method in many laboratories of using the electrical factors, I am not quite so sure that we can condemn that entirely. I know we can duplicate those conditions with sufficient accuracy to duplicate our results from patient to patient, day in and day out. The variations in the patient are so much greater than the electrical conditions, if they are carefully checked, that I think the patient himself is a much greater factor than any variation in electrical conditions.

Another point refers chiefly to Dr. Ullmann's paper, or rather the thought comes from hearing his paper and some of the other discussion which has preceded it, on this saturation method. It is interesting to see how a method will be worked out by someone and then will be forgotten for five, ten or more years, and then someone else will take it and revivify it and immediately it will spring into more or less general acceptance. That is what is taking place with the saturation method.

Kingery worked this out about 1920 working at Ann Arbor with Van Zwaluwenberg and, as a matter of fact, the idea originated with Van

Zwaluwenberg himself. Kingery deserved the credit for working it out, and it has been revivified by Dr. Pfahler.

One more point which is not directly connected with the papers but has a bearing on them, and that is that we are finding more and more that a number of converging beams to a tumor is often very much better, even at moderate voltage, than one large single field at high voltage, or any other voltage. For instance, in many tumors where, for one reason or another, nothing else can be done, we are using this method of converging beams, sometimes as many as four or six which can be worked out, depending on the part of the body, and we get results which cannot possibly be duplicated in any other way.

Dr. B. P. Widmann, Philadelphia. The enthusiasm in Dr. Pfahler's clinic about the saturation technique is due not only to the belief that the idea of keeping up an effect is good, but also because this method of treatment is applicable to all of our patients in a relative way. It gives us some scheme of treating our patients so that every day we are carrying out practically the same idea of dosage, the same system of measurements, so that while we are not treating each case exactly alike, a week or a month or a year later we are duplicating the same form of treatment on different patients and various people in the same *x*-ray department understand this system of dosage.

The reason for Dr. Ullmann's variation is very simple. Dr. Weatherwax has checked up our measurements and I believe he agrees almost exactly with Dr. Ullmann as far as I can tell from his chart. Our saturation chart was really drawn in the beginning from clinical experience and Dr. Pfahler had in mind especially the idea of keeping within safe limits. I believe any of these physical measurements that can check up the depth doses and work them out on the basis of saturation is a good idea.

Dr. R. R. Newell, San Francisco, Calif. We arrived, from clinical observation, at approximately the same rate of falling off in dosage as these other gentlemen, namely 7 per cent a day.

In regard to the order of ports in which a treatment is given to attain a maximum amount in the middle of the body, Dr. Ullmann called my attention to this a long time ago. I believe I can state a fairly simple rule to attain a maximum accumulation in the middle of the body for a given accumulation on the skin in any number of ports, and that is that you will attain the maximum number in the middle of the body for a given amount on the surface if you will treat a maximum number of ports every day. Treating all four ports every day I think, would be a close approximation to the optimum.

Dr. Ullmann's paper deals with comparatively small percentages. It seems to me impossible to demonstrate on the same patient the difference between two doses which differed by as little as 20 per cent. In other words, our clinical observation is not nearly so fine as the small quantities Dr. Ullmann is dealing with. We don't want to run too far on the theory and outstrip the possibility of any clinical check up for it.

Dr. Portmann (closing). We admit of course, that we are physicians first and that we have to treat our patients, but at the same time we should treat them with some knowledge of what we are doing. For example, the physician would not think of giving a dose of digitalis without knowing its strength. I think that we as radiologists must take into consideration the physical unit for measurement.

We talk about variations in the reactions of patients, but we can't prove them. Perhaps the variations are purely physical and have nothing to do with the organism as a metabolic chamber, if we may call it that. Dr. Wood's experimental work would indicate that there isn't as much variation in the patients as we might think. He finds that every tumor that he has seen always reacts exactly in the same way to the same amount of radiation. This would indicate at least that these tumors react to a measured physical dose without consideration at all of the animal in which it happens to live.

There is no reason for condemning the old methods of estimating dosage by means of the physical factors, except that they are not quite accurate. The best example I can think of would be the treatment of epithelioma of the lip in which the size of the lesion would have a great deal to do with the amount of radiation that the tumor might receive. We may say we give an epithelioma of the lip ten erythema doses, but this does not mean anything, as far as the intensity of the radiation is concerned, unless we know the size of the tumor. If we place lead back of the tumor, the dosage for that tumor would be much greater than if no lead were used at all.

So I think we should base all of our work, to be absolutely correct, on some physical basis. Perhaps my paper is a prophecy that some day we shall all be expressing our dosage in one international unit and we shall all understand each other.

Dr. Ullmann (closing). When Dr. Des-

jardins said that he had noted that multiple ports with converging beams appeared to have a better clinical result than a single port we wondered if that wasn't a bit of the saturation method because it is spreading the dose over a little more time.

I was afraid that many would think that any factor I might use was a standard factor which I recommended. You will note that I specifically stated that all of these factors were assumed to illustrate a principle. I do not know that 7 per cent is correct. It is the nearest average to what experimental and clinical evidence we have, and therefore was used to illustrate the principle. I am using it in my laboratory until we have something more definite. It is not a standard. I have never suggested it as such.

Another thing: I do not repeat the dose indefinitely by any means. I do not want you to think that one can figure 7 per cent or 8 per cent or 6 per cent per day loss and repeat indefinitely. It is only a figure for calculation in order to deliver a certain definite dose and repeat, we will say, once or twice, depending on the experimental and clinical evidence which may accumulate.

Dr. Desjardins emphasized the necessity for our being clinicians as well as physicists. I read a paper before this Society some years ago in which I stated that radiation dosage was a clinical and not a physical problem, and before that presented the same thought before the Radiological Society, so I do not wish to be accused of considering the physical factors of more importance than the clinical.

Regarding Dr. Newell's discussion on the number of ports, I did not recommend particularly any order of ports or their numbers. I simply said that the order of ports must be taken into consideration in calculating doses. It is a principle, not a rule.

Vol. XXI, No. 2

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

Editor: ARTHUR C. CHRISTIE, M.D.

Associate Editor: LAWRENCE REYNOLDS, M.D.

Editorial Board: JAMES T. CASE, M.D. H. K. PANCOAST, M.D. WILLIAM DUANE, PH.D.

Advisory Board for Pathology: JAMES EWING, M.D. EUGENE OPIE, M.D. A. S. WARTHIN, M.D.

Collaborating Editors: The Officers and Committee Members of the Societies of which this JOURNAL is the official organ, whose names appear on this page, are considered collaborating editors of this JOURNAL.

Foreign Collaborators: A. BÉCLÈRE, M.D., PARIS. GÖSTA FORSSELL, M.D., STOCKHOLM. G. F. HAENISCH, M.D., HAMBURG. R. LEDOUX-LEBARD, M.D., PARIS.

Publishers: PAUL B. HOEBER, INC., NEW YORK

Issued Monthly. Subscription $10.00 per year, $11.00 in Canada and $12.00 in foreign countries. Advertising rates submitted on application. Editorial office, 1909 Massachusetts Ave., N. W., Washington, D.C. Office of publication, 76 Fifth Avenue, New York.

Information of interest to all readers will be found on page ii.

Officers and Standing Committees

THE AMERICAN ROENTGEN RAY SOCIETY

President: EDWARD H. SKINNER, Kansas City, Mo.; *President-Elect:* H. M. IMBODEN, New York City; *1st Vice-President:* D. Y. KEITH, Louisville, Ky.; *2d Vice-President:* W. E. CHAMBERLAIN, San Francisco, Calif.; *Secretary:* JOHN T. MURPHY, 421 Michigan Ave., Toledo, Ohio.; *Treasurer:* WILLIAM A. EVANS, 10 Peterboro St., Detroit, Mich.; *Librarian and Historian:* H. W. DACHTLER, Toledo, Ohio.

Executive Council: G. W. GRIER, Chairman, Jenkins Arcade, Pittsburgh, Pa., W. F. MANGES, Philadelphia, Pa., L. R. SANTE, St. Louis, Mo., E. H. SKINNER, Kansas City, Mo., H. M. IMBODEN, New York City, A. C. CHRISTIE, Washington, D. C., JOHN T. MURPHY, Toledo, Ohio, WILLIAM A. EVANS, Detroit, Mich.

Committee on Laws and Public Policy: B. R. KIRKLIN, Chairman, Rochester, Minn., FRED M. HODGES, Richmond, Va., WILLIAM E. CHAMBERLAIN, San Francisco, Calif.

Safety Committee: P. M. HICKEY, Chairman, University Hospital, Ann Arbor, Mich., H. K. PANCOAST, Philadelphia, Pa., W. D. COOLIDGE, Schenectady, N. Y., A. U. DESJARDINS, Rochester, Minn., H. J. ULLMANN, Santa Barbara, Calif., B. H. NICHOLS, Cleveland, Ohio, G. E. RICHARDS, Toronto, Canada, R. R. NEWELL, San Francisco, Calif.

Committee on Scientific Exhibit: LAWRENCE REYNOLDS, Chairman, 10 Peterboro St., Detroit, Mich., IRA H. LOCKWOOD, Kansas City, Mo., LEWIS G. ALLEN, Kansas City, Mo.

Publication Committee: WILLIAM A. EVANS, Chairman, Detroit, Mich., G. W. GRIER, Pittsburgh, Pa., W. F. MANGES, Philadelphia, Pa.

Member, National Research Council: GEORGE W. HOLMES, Boston, Mass.

Editor: A. C. CHRISTIE, 1909 Massachusetts Ave N. W., Washington, D. C.; *Associate Editor,* Lawrence Reynolds, Detroit, Mich.

Editorial Board: JAMES T. CASE, H. K. PANCOAST, WM. DUANE.

Advisory Board for Pathology: JAMES EWING, EUGENE OPIE, ALDRED S. WARTHIN.

Publishers: PAUL B. HOEBER, INC., 76 Fifth Avenue, New York City.

Thirtieth Annual Meeting, 1929: To be announced.

THE AMERICAN RADIUM SOCIETY

President: EDWIN C. ERNST, Beaumont Medical Building, St. Louis, Mo.; *President-Elect:* H. H. BOWING, Rochester, Minn.; *First Vice-President:* WILLIAM P. HEALY, New York City; *Second Vice-President:* WILLIAM E. COSTOLOW, Los Angeles, Calif.; *Secretary:* G. W. GRIER, Jenkins Arcade, Pittsburgh, Pa.; *Treasurer:* ZOE A. JOHNSTON, Pittsburgh, Pa.

Executive Committee: DOUGLAS QUICK, Chairman, New York City; ALBERT SOILAND, Los Angeles, Calif.; CURTIS F. BURNAM, Baltimore, Md.

Program Committee: H. H. BOWING, Chairman, Rochester, Minn., Dr. H. J. ULLMANN, Santa Barbara, Calif., Dr. G. ALLEN ROBINSON, New York City.

Publication Committee: HENRY K. PANCOAST, Chairman, Philadelphia, Pa.; JAMES T. CASE, Battle Creek, Mich.; R. E. LOUCKS, Detroit, Mich.

Standardization Committee: G. FAILLA, Chairman, New York City; HENRY J. ULLMANN, Santa Barbara, Calif.; ROBERT B. GREENOUGH, Boston, Mass.

Fourteenth Annual Meeting: Portland, Oregon, July 8–9, 1929.

Committee on Arrangements: Dr. OTIS B. WIGHT, Chairman, Portland, Oregon, Dr. JOHN R. RANSON, Portland, Dr. ROY PAYNE, Portland, Dr. BUTLER, Portland, Dr. DORWIN PALMER, Portland.

EDITORIALS

TREATMENT OF AGRANULOCYTIC ANGINA BY IRRADIATION

IN the December, 1928, issue of the Journal Drs. Call, Gray and Hodges reported a case of agranulocytic angina with recovery after roentgen-ray treatment. They also called attention to four cases previously reported by Friedemann, all of whom recovered after roentgen irradiation.

Since then there has come to our attention another article on this subject by Friedemann[1] in which he reviews twenty-nine cases of agranulocytic angina which had come under his own observation and reports two additional cases which recovered after roentgen irradiation. Of the twenty-nine cases reported by Friedemann five were in men and twenty-four in women which is in accordance with the general observation that the disease preponderantly involves women. It was not observed in childhood and in only one case under twenty years of age, otherwise it occurred quite uniformly at all ages. A connection with other preceding diseases could not be established from the histories.

The disease in most cases comes on acutely, the first symptoms being due to the angina. The duration varies somewhat but in about half of Friedemann's cases it terminated fatally in the course of eight days while it was protracted in others from twenty-four up to sixty days.

The disease of the tonsils is usually a necrotic ulceration but in some cases only a follicular angina was observed and in others only a greasy white coating. In the beginning of the disease the appearance simulates diphtheria. The early dysphagia is always striking. Besides the angina other necroses were frequently observed: in two cases there was extensive destruction of the mucous membrane of the larynx, in two cases necrosis of the mucosa of the vagina and vulva, in three cases ulcers in the pharynx and anus, in two cases severe ulcerous stomatitis and in one case necrosis of the superior maxilla. Two cases in which the tonsils remained free from disease and the mucous membranes of the cheek, tongue, rectum and bladder were involved were especially interesting because they may be regarded as proof that the tonsils are not the primary seat of the disease.

The lesions of the tonsils and of the mucous membrane elsewhere are not characteristic and it is only the blood picture which is diagnostic. There is a pronounced leucopenia and the granulocytes are either absent or reduced to exceedingly low percentages. The absolute number of lymphocytes is usually decreased but it may be normal. The degree of anemia is not so severe as that seen in the aleucemic forms of the myeloses and lymphadenoses but the erythrocytes are usually reduced, the count being commonly from 2,500,000 to 3,500,000. The blood platelets retain a normal count.

The autopsy findings throw little light on the cause of the disease. The spleen, lymphatic glands and liver showed no macroscopic or microscopic changes from normal.

In only six cases were bacteria demonstrated in the blood. The result of Friedemann's bacteriological examinations contradicts the idea that the agranulocytosis is only a special reaction type of severe septic disease. He believes the sepsis to be a secondary manifestation.

The prognosis in this disease has always been bad. Friedemann reports two cases of recovery out of twenty-four observed by him, eliminating the five who recovered after roentgen therapy. He states that about the same mortality is observed in cases reported by others (43 deaths in 47 cases). The mortality rate is therefore about 91.6 per cent.

On the strength of his own observations Friedemann draws the following conclusions as to the nature of the disease: (1) The transformation of the blood picture must be considered an essential sign for the diagnosis of agranulocytosis. It consists in

[1] *Ztschr. f. klin. Med.*, 1928, cviii, 54–56.

a marked decrease of the granular leucocytes, a complete disappearance need not always occur but the relative number of granulocytes must be reduced to a low percentage. The absolute number of lymphocytes is also markedly decreased. An anemia of a moderate degree is usually present.

(2) Necroses of the mucous membranes are always present if the disease has been present for some time. In the majority of cases they are localized in the tonsils, but necroses may occur in the cheek, gums, tongue, nose, vulva, vagina, intestines and bladder. In one case necroses of the skin were observed. (3) Icterus is a symptom which sometimes occurs but it is of secondary importance. (4) The disease involves preponderantly the female sex. (5) The prognosis is unfavorable. (6) The etiology is not understood but the author does not believe it to be due primarily to infection. The angina, he thinks, is a secondary manifestation of a disease of the hematopoietic organs.

Friedemann refers to four cases of this disease which he had previously reported and which were cured by irradiation, and in the paper here under review he reports two additional cases cured by the same treatment. These, with the case reported by Call, Gray and Hodges in the December issue of this Journal bring the total so treated up to seven. The prompt improvement in the blood picture and throat manifestations and the final return to normal in all of the seven cases treated by irradiation indicates very strongly that the treatment was responsible for the results.

Renewed attention is called to this method of treatment so that care may be taken to examine the blood in all cases of angina and that roentgen treatment over the long bones may be tried in a sufficient number of cases to demonstrate whether or not the results so far obtained are constant. It should be kept in mind that the effect is presumably upon the hematopoietic organs and that little good can be expected if a secondary general sepsis has occurred.

FRANK BUTLER GRANGER, M.D.

Dr. Frank Butler Granger, died at his home in Boston, October 23, 1928, of metastatic carcinoma of the liver.

Dr. Granger was one of the first physicians of the regular school in New England to practice the treatment of disease by electrical methods. He long stood alone convinced of the truth that lay in physical therapy, weathered the sorties of the charlatans into his chosen field and lived to see it recognized as an important specialty in medicine.

His last message to the profession was given to the American Academy of Physical Therapy in session in New York City a few days before his death, and closed with the words, "Adopt no holier than thou attitude."

It epitomizes his kindliness of thought, which was a characteristic of his every act. Only those who were close to him in his latter days will grasp the full strength of his character. With the knowledge that he was doomed, frightfully distressed, he could smile and listen with patience to those with ailments trifling compared with his own and certainly less cheerfully borne.

He was a graduate of Harvard University Medical School, Boston, 1902; member of the Council on Physical Therapy, American Medical Association; lecturer on physical therapeutics, 1906–1910, and assistant professor of physical therapy, 1927–1928, Tufts College Medical School; instructor, Harvard Medical School, courses for graduates, 1911–1925. He entered the medical corps of the U. S. Army during the World War as a captain and was discharged in 1919 as a lieutenant colonel. He was organizer and director of the department of physical therapy, division of physical reconstruction, Surgeon General's office, Washington; at one time counselor of the medical council of the U. S. Veterans' Bureau; past president of the American Academy of Physiotherapy and the American Electro-Therapeutic Association; member of the American Roentgen Ray Society.

He was appointed electro-therapeutist to the Boston City Hospital, April 22, 1908, and organized the department of physical therapeutics which he developed to a plane second to none in this country.

FREDERICK W. O'BRIEN.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this column solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. John T. Murphy, 421 Michigan Ave., Toledo, Ohio.
Thirtieth annual meeting, 1929: To be announced.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Dr. Albert Soiland, 1407 S. Hope St., Los Angeles, Calif.
Annual meeting, Portland, Oregon, 1929.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Fred M. Hodges, 801 West Grace St., Richmond, Va.
Annual meeting, Portland, Oregon, July 8–12, 1929.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Walter T. Bronson, Northwestern Univ. Med. School, Chicago, Ill.
Fifteenth annual session, 1929: To be announced.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL SOCIETY
Secretary, Dr. Lowell S. Goin, Wilshire Medical Building, Los Angeles.
Meets on the third Wednesday of each month at the California Hospital.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. W. S. Lawrence, Medical Arts Bldg., Memphis, Tenn.

BUFFALO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph S. Gian-Franceschi, 610 Niagara St.
Meets second Monday of each month except during the summer months, the place of meeting to be selected by the host.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. W. T. Bronson, Englewood Hospital.
Meets monthly on second Thursday from October to May (except during month of December) at Virginia Hotel. Dinner at 6 P.M., scientific session at 8 P.M.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. Harry L. Farmer, 2930 Prospect Ave.
Meetings are held at 6 o'clock at the University Club, on the fourth Monday evening of each month from September to April, inclusive.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital.
Meets monthly on second Friday from October to May, at Wayne County Medical Society Building.

CENTRAL ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. H. C. Kariher, Decatur, Illinois.
Regular meetings held quarterly.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. Carl S. Oakman, Muncie, Indiana.
Annual meeting each February 22 in Indianapolis. Next meeting in Indianapolis, February, 1929.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. J. E. Habbe, 221 Wisconsin Ave., Milwaukee.
Meets first Friday in October, December, February and April.
Place of meeting designated by the president.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. M. C. Sosman, Peter Bent Brigham Hospital, Boston, Mass.
Meets monthly on third Friday, Boston Medical Library.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. E. Everett Rowell, 200 West 59th St.
Meets monthly on third Monday, New York Academy of Medicine.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. D. S. Childs, 316 Gurney Bldg., Syracuse, N. Y. Three meetings a year—April, August and November.

PACIFIC COAST ROENTGEN RAY SOCIETY
Secretary, Dr. Harold B. Thompson, Seattle, Wash.
Two meetings a year.

ROENTGEN RAY SOCIETY OF CENTRAL PENNSYLVANIA
Secretary, Dr. W. E. Reiley, Clearfield, Penna.
Two meetings a year, April and October.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary Dr. John T. Farrell, Jr., 235 S. 15th St., Philadelphia.
Meets monthly on first Thursday evening, Pennsylvania Hospital.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. James M. Flynn, 282 Alexander St.
Meets monthly on the first Friday evening at 7.45 at the Rochester Medical Association Building.

ST. LOUIS ROENTGEN CLUB
Secretary-Treasurer, Dr. L. R. Sante, Metropolitan Building.
Meets first week of each month. Time and place of meetings designated by president.

TEXAS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. C. P. Harris, Houston, Texas.
Meets annually one day preceding the meeting of the Texas State Medical Association.

VIRGINIA ROENTGEN RAY CLUB
Secretary, Dr. Wright Clarkson, 205 S. Sycamore St., Petersburg, Va.
Annual meeting, Charlottesville, Va., Oct. 18, 1929.

BRITISH EMPIRE

BRITISH INSTITUTE OF RADIOLOGY INCORPORATED WITH THE RÖNTGEN SOCIETY
Director, Dr. John Muir, 32 Welbeck St., London, W. 1.

* Secretaries of societies not here listed are requested to send the necessary information to the editor.

Meets on the third Thursday of each month, from November to June inclusive, at 8.15 P.M., at 32 Welbeck St., London, W. 1., or as advertised.

ELECTRO-THERAPEUTIC SECTION OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)

Meets on the third Friday of each month during the winter at 8. 30 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

RADIOLOGICAL SECTION, AUSTRALASIAN MEDICAL CONGRESS

Secretary, Dr. C. A. Anderson, The Hospital, Dunedin.

RADIOLOGICAL SECTION OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION

Secretary, Dr. Colin Macdonald, Lister House, 61 Collins St., Melbourne, Australia.

Meets monthly at Melbourne during the winter.

SECTION ON RADIOLOGY, CANADIAN MEDICAL ASSOCIATION

Secretary, Dr. A. H. Rolph, 160 St. George St., Toronto, Ont.

RADIOLOGICAL SECTION, NEW ZEALAND BRITISH MEDICAL ASSOCIATION

Secretary, Dr. P. C. Fenwick, The Hospital, Christchurch.

Meets annually.

Continental Europe

BELGIAN SOCIETY OF ROENTGENOLOGY

Secretary, Dr. J. Boine, Avenue des Alliés, 134, Louvain (Belgium).

Meets monthly on second Sunday at d'Egmonds Palace, Brussels, except in the summertime.

SOCIETE DE RADIOLOGIE MÉDICALE DE FRANCE

Meets monthly on second Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

SOCIETE FRANCAISE D'ELECTROTHÉRAPIE ET DE RADIOLOGIE MÉDICALE

Meets monthly on fourth Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

ASSOCIATION OF GERMAN ROENTGENOLOGISTS AND RADIOLOGISTS IN CZECHO-SLOVAKIA

Secretary, Dr. Walter Altschul, German University, Prague, 11/52.

DEUTSCHE RÖNTGEN-GESELLSCHAFT (GESELLSCHAFT FÜR RÖNTGENKUNDE UND STRAHLENFORSCHUNG)

Meets annually in April, alternating one year in Berlin, one year in some other German city. Meets in addition every two years with the Gesellschaft deutscher Naturforscher und Aerzte.

Permanent secretary, Professor Dr. Haenisch, Klopstockstrasse 10, Hamburg, Germany.

SUD- UND WESTDEUTSCHE RÖNTGENGESELLSCHAFT

Meets annually in different cities.

NORD- UND OSTDEUTSCHE RÖNTGENGESELLSCHAFT

Meets annually in different cities.

DUTCH SOCIETY OF ELECTROLOGY AND ROENTGENOLOGY

Holds two meetings a year in Amsterdam, one in the Spring, and one in the Fall.

SOCIETA ITALIANA RADIOLOGIA MEDICA

Secretary, Professor M. Ponzio, University of Turin, Turin.

ALL-RUSSIAN ROENTGEN RAY ASSOCIATION, LENINGRAD, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.

Secretaries, Drs. S. A. Reinberg and S. G. Simonson.

Meets annually.

Sixth Annual Meeting, 1929, to be announced.

LENINGRAD ROENTGEN RAY SOCIETY

Secretaries, Drs. S. G. Simonson and G. A. Gusterin.

Meets monthly on the first Monday at 8 o'clock in the State Institute of Roentgenology and Radiology, Leningrad.

MOSCOW ROENTGEN RAY SOCIETY

Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky.

Meets monthly on the first Monday at 8 o'clock, the place of meeting being selected by the Society.

POLISH SOCIETY OF RADIOLOGY

Secretary, Dr. A. Elektorowicz, 19 Hoza St., Warsaw.

Meets annually.

WARSAW SECTION, POLISH SOCIETY OF RADIOLOGY

Secretary, Dr. B. Krynski, 11 Zielna St.

Meets once a month except in the summertime.

SCANDINAVIAN ROENTGEN SOCIETIES

The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association. Each of the following societies, with the exception of the Denmark Society, meets every second month except in the summertime:

SOCIETY OF MEDICAL RADIOLOGY OF SWEDEN

Meets in Stockholm.

SOCIETY OF MEDICAL RADIOLOGY IN NORWAY

Meets in Oslo.

SOCIETY OF MEDICAL RADIOLOGY IN DENMARK

Secretary, Dr. H. Scheuermann, Copenhagen

Meets on the second Wednesday of each month from October to July in Copenhagen, at 8 o'clock in the State Institute of Roentgenology.

SOCIETY OF MEDICAL RADIOLOGY IN FINLAND

Meets in Helsingfors.

VIENNA SOCIETY OF ROENTGENOLOGY

Secretary, Professor Holzknecht, Vienna, IX, General Hospital.

Meets on the first Tuesday of each month from October to July.

VIENNA LETTER

(From our Special Correspondent)

THE ESTABLISHMENT OF A CANCER COMMISSION AS A PART OF THE LEAGUE OF NATIONS

The League of Nations has selected Munich, Germany, as a meeting place for the Radiological Committee which will deal especially with radiation treatment of carcinoma. Munich was selected as a meeting place for the committee because

it possesses one of the oldest gynecological clinics and has the most comprehensive statistics relating to the radiation treatment of carcinoma. The committee consists of the following members: Dr. Comyns-Berkely, director of the Middlesex Hospital, London; Dr. Lane-Claypon, referee in the British Ministry of Sanitation; Professor Regaud, director of the Radium Institute in Paris; Dr. Lacassagne of the same Institute; Professor Döderlein, director of the Gynecological Clinic in Munich; Professor Boltz, radiologist to the same Institute; Professor Lahm, director of the radiological laboratory of the Gynecological Clinic in Rome, Italy; Professor Forssell, director of the Radium Institute in Stockholm, Sweden; Professor Heyman of the same Institute; Professor Roy, director of the Gynecological Clinic of Amsterdam, Holland.

The first meeting of the committee was held on December 11, 1928. At this meeting Professor Döderlein in his inaugural address stated that he is opposed to the operative treatment of carcinoma of the female pelvic organs. His experiences gained from a study of the vast material of the Munich Gynecological Clinic has led him to the conclusion that radiation treatment of carcinoma of the female pelvic organs is far more efficacious than other methods. The reports of the results of radiation treatment in Paris and Stockholm differ somewhat. This Professor Döderlein attributes to the fact that in Paris the gynecological and radiological departments are separate, while in Stockholm the two departments are more closely associated, which permits of a more careful study of the individual patient.

Dr. Tomanek, the secretary of the Cancer Commission, member of the Health Section of the League of Nations, outlined the scope and purposes of the commission as follows: First, the commission desires to make a comparative study of the methods and the results obtained in the treatment of uterine carcinoma. This particular disease has been selected because this type of carcinoma, in contradistinction to other forms, has been for a long time the subject of a very wide and intensive research. Secondly, the commission proposes to make a very comprehensive study of the propagation and the etiology of so-called occupational cancers. It is the purpose of the commission to pay particular attention to carcinomas of the lung occurring in workers in the cobalt mines in Schneeberg. In Czechoslovakia a special commission has been appointed for this type of investigation and also in the Joachimsthal radium mines where the working conditions are about the same as those obtaining at the other places. Thirdly, the commission proposes to send questionnaires to all cancer research workers throughout the world. These questionnaires will detail the different carcinoma problems under consideration which may be studied with the help of the League of Nations and which give promise of early and successful solution. Dr. Tomanek took occasion to remark that the League of Nations lays particular stress not upon theoretical investigations but upon the more practical handling of the fight against cancer. The commission further proposes to give lectures, exhibition of instructive cases and to acquaint the members of the commission with the organization and statistics of cancer treatment in all the prominent clinics throughout the world.

THE DISCOVERY OF THE PROBABLE CAUSATIVE AGENT OF LEUCEMIA

At a recent meeting of the Vienna Roentgen Society, Dr. Franz Freund read a very interesting paper on a case of leucemia of the skin in which he studied the changes in the skin after treatment with roentgen rays. In microscopic preparations he found peculiar cells which he calls protozoa, an observation which has been corroborated by other observers. He was somewhat hesitant in stating dogmatically that these protozoic cells were definitely the cause of leucemic affections. However, the infectious theory of leucemia is not a new one. More than twenty years ago Professor Löwit of Innsbruck stated that leucemia is of infectious origin. His view did not find general acceptance, however, particularly so in view of the fact that Professor Türk of Vienna demonstrated that the forms in the microscopic preparations

of Professor Löwit were not protozoa. On the other hand, Professor Pappenheim of Germany stated it to be his opinion that leucemia is an infectious disease, the carriers of which may be revealed in some cells of the blood. This view has not received general acceptance. It is gratifying that the recent findings of Dr. Freund were observed during roentgen-ray treatment which may ultimately lead to the final solution of the problem of the infectious theory of leucemia and also to its effective treatment.

THE TREATMENT OF EXUDATIVE DIATHESIS BY THE IRRADIATION OF THE THYMUS GLAND

At a recent meeting of the Medical Society, Dr. J. Jenö Vas stated that he had made a roentgen-ray examination of all children admitted to the Children's Clinic and had found a definitely enlarged supracardiac shadow in almost all the cases of exudative diathesis, which group of diseases is most closely associated with so-called status thymicolymphaticus. He systematically applied deep roentgen-ray treatment over the thymic area in 36 of these children who were suffering from eczema, prurigo and urticaria pigmentosa. His results were variable. However, the favorable results achieved in certain cases prompts him to suggest this method of treatment in all such cases where this type of diathesis is refractory to hitherto employed dietetic and external methods of treatment. According to his opinion the reduction of the function of the thymus is conducive to a loss of alkali in the organism and this favorably influences the improvement seen in these exudative diatheses.

THE ROENTGEN DIAGNOSIS OF INTRAUTERINE DEATH OF THE FETUS

At a recent meeting of the University Scientific Association of Budapest Dr. Francis Szellö called attention to the fact that intrauterine fetal death cannot always be clinically diagnosed and in such instances considerable assistance may be had from the roentgen-ray examination of the mother. When fetal death has occurred there is shown a flattening of the roof of the skull, with an asymmetry and collapse of the whole skull as a consequence of the post-mortem shrinking of the intracranial contents. There is also an appreciable bending of the spinal column with an overriding of the edges of the bones upon one another. Spalding was the first to call attention to this method of examination; others, chiefly American authors, have published their views and observations on intrauterine fetal death. Dr. Szellö chiefly emphasized the fact that the roentgen examination is only of assistance before the head is engaged and before the actual onset of labor.

INFLUENCING THE SIZE OF THE HEART BY ATROPINE, ADRENALIN AND AMYL NITRITE

At a recent meeting of the Vienna Medical Society Dr. D. Scherf reported a series of experiments which he had carried on in his clinic on the measurements of the size and shape of the cardiac shadow after the introduction of varying solutions of atropine, adrenalin and the use of amyl nitrite. He determined the size of the heart roentgenographically by the method of Gött and Rosenthal, with certain modifications. His experiments led him to the following conclusions: Intravenous injections of 0.002 gm. of atropine in healthy subjects caused, in the stage of acceleration, a diminution in the systolic and diastolic heart diameters, that is, a diminution of the size of the heart. The diminution in general is the more pronounced with the increase of the pulse rate. The diminution of the diameter of the heart as determined roentgenkymographically was considerably less than that established by Moritz orthodiagraphically after subcutaneous injection of 0.002 gm. atropine. The normally expected change in the systolic size of the margins of the heart shadow, and accordingly that of the beating volume could not be observed and confirmed. The intravenous injection of 0.05 c.c. of a 1 per cent solution of adrenalin never produces in a normal subject any increase in the size of the heart. However, in a few of the cases a definite diminution in the kymographic curve could be noted. An

increase of the size could not be observed even when the blood pressure rose over 200 mm. mercury. This may be explained on the ground that the positive inotropic action which is conducive to the diminution of the size of the heart and a rise of blood pressure has the effect of increasing the size of the heart, counterbalanced each other and in exceptional cases one of them —the former rather—predominated. In some cases besides the diminution of the diameter of the heart there was also a diminution of the excursion of the heart, a fact which points in all probability to an actual diminution in the beating volume. It could also be observed that upon the inhalation of amyl nitrite the size of the cardiac shadow definitely diminished.

A CASE OF INFLAMMATORY TUMOR FORMATION IN THE TRANSVERSE MESOCOLON

At a recent meeting of the Vienna Roentgen Society Dr. Pape exhibited a case in which an inflammatory tumor formation was masked by the clinical picture of stenosis of the rectum. The patient three weeks prior to the roentgen examination had been operated on for inguinal hernia and meanwhile had lost 14 kilos in weight. He had considerable pain in the abdomen and repeated roentgen examination revealed the presence of a persistent filling defect 10 cm. long in the region of the transverse colon. The form and extent of this defect varied at different examinations, and upon palpation over this area there was a feeling of resistance corresponding to that of a contracted colon. The roentgen diagnosis was a spastic colon probably secondary to a colitis with probable organic changes in the walls of the colon. At operation there was exposed an extensive inflammatory mass in the transverse mesocolon as well as in the retroperitoneal connective tissue, with extensive peritoneal adhesions. These changes gave the roentgen picture which could not be differentiated from a filling defect caused by a tumor.

THE EFFECT OF ULTRA-SHORT RADIO WAVES UPON THE TUBERCLE BACILLUS

It is no recent observation that ultra-short radio waves have a definite effect upon living organisms. Professor Esau of the University of Jena, Germany, carried on extensive experiments with these short waves, ranging from 2 to 5 meters, and he found that insects upon being exposed to these rays were killed in a few seconds. The deleterious effect of these rays is noticeable not only upon insects but upon bacteria, as for instance, the tubercle bacillus. According to Professor Esau the ultra-short waves form a powerful and hitherto unknown weapon against bacteria. He states that this is not only a hypothesis, but a fact borne out by a series of experiments. He exposed a culture of tubercle bacilli to the action of the ultra-short waves and found that shortly afterward they had entirely lost their activity. After this observation he inoculated a series of mice with tubercle bacilli and tried the effect of short waves upon these mice. Those which were exposed to the ultra-short waves were resistant to the inoculations while all of the non-exposed mice contracted tuberculosis and showed every manifestation of the disease. Professor Esau has not yet extended his experiments to humans. However he exposed to the rays sluggish wounds and ulcers which resisted all kinds of treatment and observed that although other methods of treatment had failed to heal the obstinate wounds, under the action of the ultra-short waves they were rapidly healing, new healthy granulation tissue and afterwards normal epithelium having been formed. Professor Esau will continue his experiments on human subjects, and further observations will prove whether the ultra-short waves are really of such bactericidal action as they seemed to be in the case of mice.

COMMITTEE FOR INVESTIGATION OF X-RAY TUBE SITUATION

The committee to study the situation with regard to roentgen tubes has been organized by Dr. David R. Bowen, chairman, as follows: Dr. E. C. Ernst, St. Louis; Dr. G. W. Grier, Pittsburgh, Dr. I. H. Lockwood, Kansas City, and Dr. E. A. Pohle, Madison.

Vol. XXI, No. 2

DEPARTMENT OF TECHNIQUE

Department Editor: David R. Bowen, M.D., Pennsylvania Hospital, Philadelphia, Pa.

In this department will be published from time to time, as material is available, suggestions for improvement in technique.

ROENTGEN TECHNIQUE WITH TUBE VOLTAGE AS THE VARIABLE

BY RALSTON PATERSON, M.D.

Division of Roentgenology, University of Chicago

CHICAGO, ILLINOIS

IN the taking of any roentgenogram there are, apart from the question of screens, grids and filters, four main variables: (1) the current through the tube; (2) the distance between tube and film; (3) the time of exposure, and (4) the voltage applied to the tube (measured in kilovolts).

For any type of body part, tube current is relatively fixed, depending upon the focus of the tube. Fine focus tubes carry only a small current and therefore require longer exposure time, but they give excellent detail. Broader focus tubes carry heavy currents if desired but give poorer detail. In practice one uses the smallest focus tube which will keep exposure time within reasonable limits for the part roentgenographed when the tube is made to carry its greatest allowable current.

Tube distance ought to be the greatest that can be used without undue prolongation of exposure time because with increasing distance the distortion of the shadow becomes less. When using a grid (Potter-Bucky diaphragm) the tube distance is fixed by the construction of the grid.

In the earlier days of roentgenology it was the custom to control photographic effect for varying thickness of part by varying exposure time while keeping tube current, distance and voltage practically fixed. Recently the introduction of fine autotransformer control machines has made it common practice to vary both tube voltage and exposure time. It was first suggested to me by Mr. W. W. Mowry that for particular groups of body parts it was possible to obtain adequate control of exposure by varying only one of the factors, namely tube voltage, with all of the others fixed.

Time and the other factors must vary from group to group. For instance, some parts must be roentgenographed using broader focus tubes with the shortest possible exposure time to avoid the effects of respiratory movement, while other parts can be sufficiently well immobilized to allow the use of finer focus tubes and consequently longer exposure. Thicker parts must be roentgenographed on the grid using intensifying screens, while other parts not requiring the grid can be taken using films in cardboard holders. All parts of the body commonly roentgenographed can be placed in nine groups: 1. Extremities regular. 2. Extremities rapid technique. 3. Skull and trunk bones regular. 4. Special skull technique. 5. Trunk soft parts. 6. Chest regular. 7. Chest special. 8. Stomach. 9. Trunk bones special low voltage technique. Within each of these groups it is possible to fix time, distance, screens, and vary only kilovoltage to compensate for varying thickness of the part. The method is of particular advantage when using the grid because grid speed, when once set, need not be altered from patient to patient as is necessary when time is varied. Except for special examinations such as those following barium meal or other contrast medium, more effective results are obtained by lower rather than higher kilovoltages even though this entails prolongation of exposure time.

With highly trained and experienced technicians it is possible to get good results by mere personal estimation of the patient's density yet with the ordinary technician, or with the semitrained technician, I believe that better results are obtained by actual measurement of the

thickness of the part being roentgenographed. While not essential in small parts such as ankles or knees, in spines or chests where the variation from person to person is so wide, measuring of the thickness of the part to be roentgenographed gives more uniform results than the mere estimation of the patient as "medium," "light," or "heavy." Using tube voltage as a single variable this becomes easy as one has only to chart the measured thickness of the part against a suitable kilovoltage scale.

By grouping the parts to be roentgenographed thus, it is possible to draw up a series of charts by which the exposure technique may be determined merely by measuring the part thickness and using the tube voltage connected thereto with with the one standard technique for that type of part. When establishing technique with a new plant it is advisable to have a separate chart for each of the groups and to experiment with that until it duplicates consistently. As a general average, from 7 to 9 actual peak kilovolts are added or subtracted for every inch of increased or lessened thickness of part up to tube limits. After each group has been tested out it is possible by maneuvering the time or the distance, somewhat, to amalgamate several charts into one. Figures 1 and 2 show charts for the technique which we have had in use for some time and have found effective.

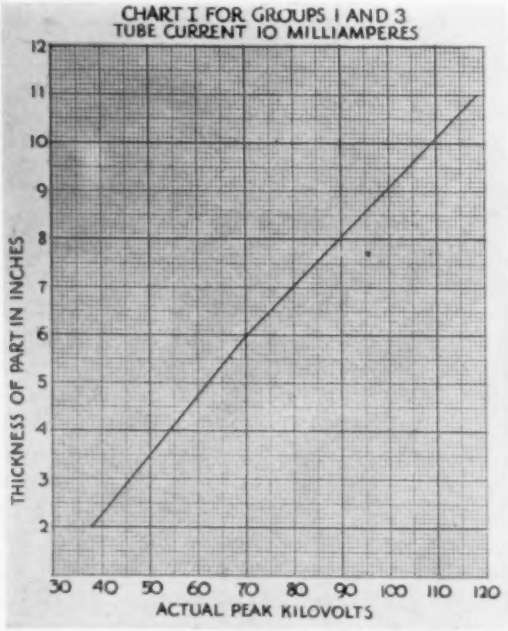


Fig. 1. Chart I is a graph showing relationship between actual peak kilovoltage and thickness of part for groups 1 and 3. It illustrates a preliminary step in the preparation of Chart III.

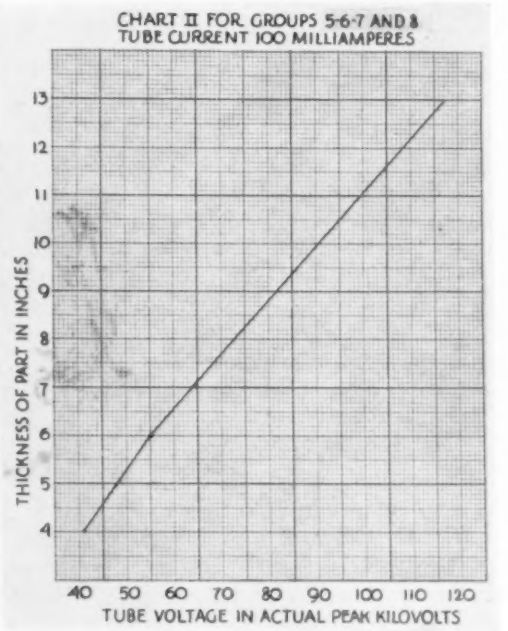


Fig. 2. Chart II. Same as Chart I except that it is for use with groups 5, 6, 7 and 8.

The technique in each group is as follows:

Group 1.

Extremities Regular. These are done with film in cardboard holders, without screens and without grid; diaphragming as necessary. At 22 in. distance and 20 ma. the exposure is 6 seconds. It is safe to use the 10 ma. tube for this because the voltage is very low. If aluminum stereoscopic tunnels are used the time must be increased to compensate for absorption in the aluminum. No account is taken of the part roentgenographed: whether it be a finger or a shoulder one merely measures the thickness of the part and uses the kilovoltage indicated on the chart. If necessary, distance may be increased to 28 in. and the time doubled.

Group 2.

Extremities, Rapid Technique. In the

roentgenographing of the extremities of children or unconscious patients, it is frequently impossible to immobilize the part, and exposure time must therefore be as short as possible. To accomplish this we use the 100 ma. tube operating at

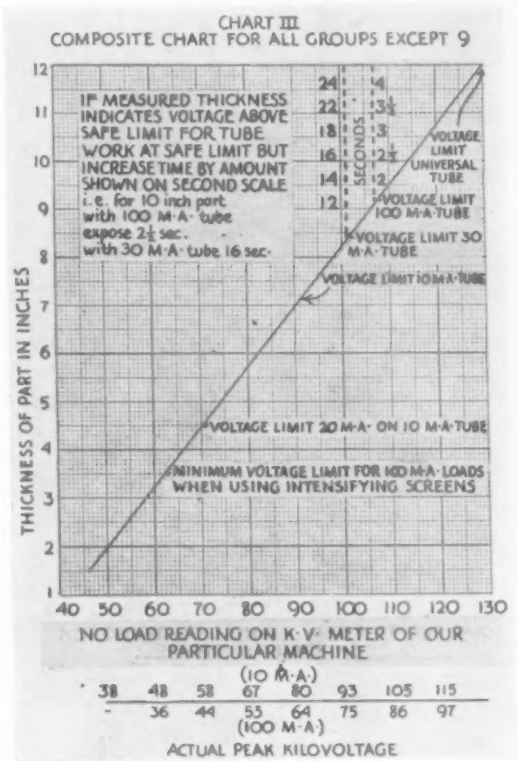


CHART III
COMPOSITE CHART FOR ALL GROUPS EXCEPT 9
IF MEASURED THICKNESS INDICATES VOLTAGE ABOVE SAFE LIMIT FOR TUBE WORK AT SAFE LIMIT BUT INCREASE TIME BY AMOUNT SHOWN ON SECOND SCALE i.e. for 10 inch part with 100 M·A· tube expose 2¼ sec. with 30 M·A· tube 16 sec.
SECONDS
VOLTAGE LIMIT UNIVERSAL TUBE
VOLTAGE LIMIT 100 M·A·TUBE
VOLTAGE LIMIT 30 M·A·TUBE
VOLTAGE LIMIT 10 M·A·TUBE
VOLTAGE LIMIT 20 M·A· ON 10 M·A·TUBE
MINIMUM VOLTAGE LIMIT FOR 100 M·A·LOADS WHEN USING INTENSIFYING SCREENS
THICKNESS OF PART IN INCHES
NO LOAD READING ON K·V· METER OF OUR PARTICULAR MACHINE
(10 M·A·)
(100 M·A·)
ACTUAL PEAK KILOVOLTAGE

FIG. 3. Chart III. A composite graph for use with all groups of parts except group 9. The no-load kilovoltmeter readings are valid only for the particular machine used by us but the values for actual peak kilovoltage are generally applicable.

The minimum voltage limit for 100 ma. loads must be observed when using intensifying screens because with voltages lower than 38 (actual peak) the front screen absorbs so much radiation that its presence retards rather than accelerates the exposure.

full capacity; film between double intensifying screens; an exposure time of ¼ sec. and the same no-load kilovolt reading as with the regular 20 ma. technique. The actual peak kilovoltage is considerably lower, of course, and the milliampere-seconds only 25 as against 120, but this is compensated for by the screens, and the resulting film is of about the same photographic density as one made by the regular technique.

Group 3.

Trunk Bones. To avoid the effect of scattering, all deeper lying parts of the skeleton must be taken on the grid, which automatically fixes the distance and entails the use of screens. The group includes skull,

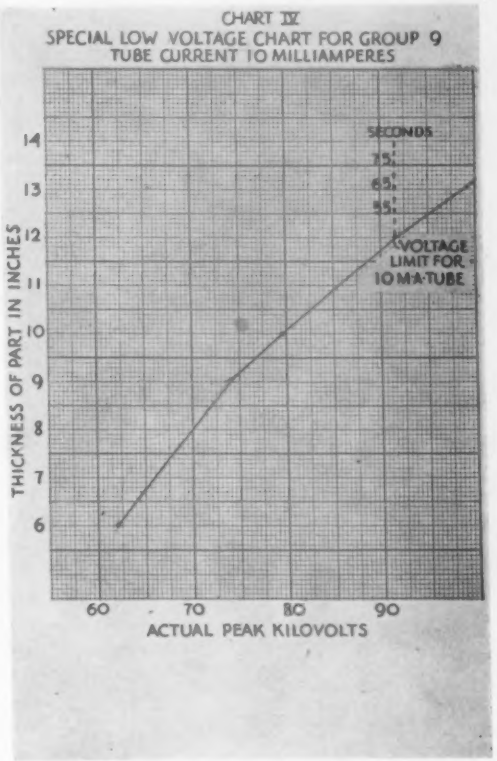


FIG. 4. Chart IV. Low voltage chart for use with group 9. Values are in actual peak kilovolts. Side C of the measuring ruler (Fig. 6) is calibrated in no-load readings converted from the values of this graph.

neck, large shoulders, large knees, spine, pelvis, ribs, etc. The 10 ma. tube is used to get fineness of detail. Using a 30 in. radius grid, exposure time is 10 sec.

Group 4.

Special Technique for Skull. We have a special skull table equipped with a small grid and having a fixed 25 in. target-film distance. For films with radiation directed through from front to back or the reverse the rays have to pass through an unusually large amount of bone. Because of this a current of 30 ma. is used. Time is 4 sec. The lateral view is done with 20 ma.; the other factors remain unchanged.

Group 5.

Body Soft Parts. The group includes kidney, gall-bladder, colon and pregnant uterus. Because of the thickness of the parts, it is necessary to use the grid, but in all of these examinations it is important to make the film during suspended respiration, so that speed is essential. Because of this we use film in double screen cassettes and a hundred-milliampere tube operating at 100 ma. The time is 1¼ sec., and the voltage is that indicated on the thickness chart.

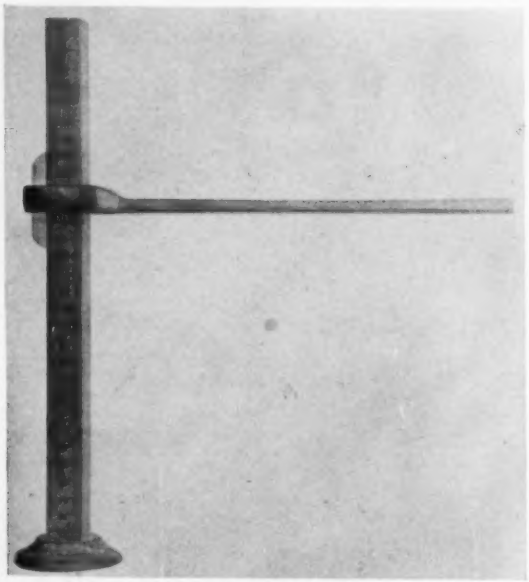


Fig. 5. Measuring ruler assembled for use. The side shown is A of Figure 6. In roentgenographing any part of the body (except group 9) the operator consults Table 1 for current, time and distance values, measures the thickness of the part with this ruler and reads off directly the no-load kilovolt reading that should be used. For a hand this will be as low as 42 on the meter and for some chests as high as 140.

Groups 6 and 7.

Chest. The regular chest film, made with the patient facing the film, is done at a distance of 72 in.; a current of 100 ma., and an exposure time of ¼ sec. In a rigid application of the voltage chart oblique and lateral chests should also be done at 72 in. with a voltage indicated by the thickness of the chest in the oblique and lateral diameter. In practice, however, this would run the voltage above the safe limit for the tube and so we reduce the distance to 50 in. for obliques and 35 in. for laterals, use 100 ma., but use the voltage indicated by the thickness of the chest in the regular posteroanterior position. When patients are too ill to stand or sit in front of the chest film changer and must be roentgenographed lying on their backs in bed it is not convenient to use a target-film distance of 72 in. In such cases a dis-

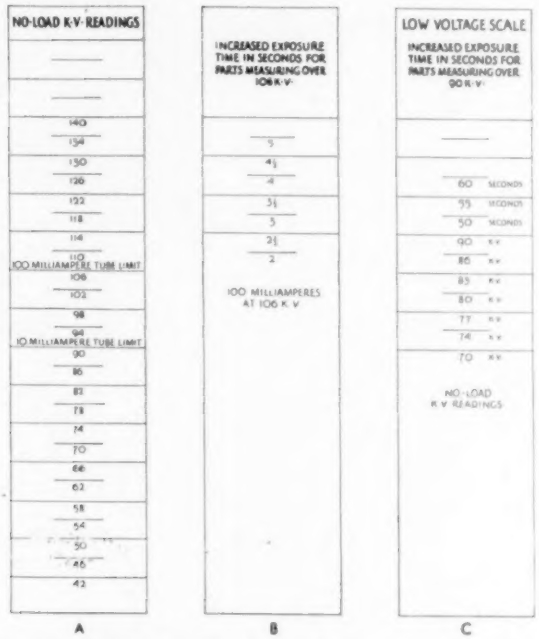


Fig. 6. Sketch of calibration scales on measuring ruler. (Reduced four-fifths.) All voltage values are in no-load kilovoltmeter readings on a 14 in. Kelley-Koett double disc X-ray machine.

Side A embodies the values of Chart III and is used with all groups of parts except group 9.

Side B is used to determine the necessary time of exposure in cases where the thickness of the part would indicate a voltage higher than the safe limit of the 100 ma. tube. For instance, in roentgenographing the gall-bladder of a very stout patient it may be found that the body measures 134 kv. (about 32 cm.), which with a load of 100 ma. means an actual peak kilovoltage of about 112, well above the limit of safety. In such cases the operator holds the slider at 134, turns the ruler to side B and notes that if the voltage is held at the safe limit of 106 kv. (no-load) exposure time must be increased to 5 sec. No provision is made for lengthening exposure when thick parts indicate a voltage greater than 90 (no-load) for the 10 ma. tube because in practice such parts are done with the low voltage scale (side C).

Side C. Low voltage scale for use with group 9. Values in no-load kilovolt readings for our machine; obtained by converting peak kilovolt values from Chart IV. For parts thicker than 90 kv. no-load (25 cm.) the ruler indicates an increase of exposure time with voltage fixed at 90.

tance of 30 in. is used and the exposure time is reduced to 1/10 sec.

Group 8.

Stomach. In roentgenographing the barium-filled stomach the problem is practically the same as in the parts in group 5 except that speed is such an important factor that we prefer to work without the grid and thus be able to reduce exposure time to ½ sec.

Group 9.

Low Voltage Technique for Spine and Pelvis. Mr. Glen Files first demonstrated to me the advantages of the 10 ma. tube and extremely low voltage in all cases of deep-lying bony parts where it is possible to obtain good strap immobilization for the long exposures necessary. Films made in this manner show excellent contrast. The same principle of voltage control can be applied. Figure 4 shows a chart of this technique.

When the kilovoltage scale runs beyond the safety limit of the tube (on our charts this occurs with very stout patients), one uses the maximum allowable voltage of the tube and increases exposure time.

Charts I and II (Figs. 1 and 2) are in actual peak kilovolts as calibrated by sphere gap and are applicable to any

TABLE 1

Current, time, and distance values for the nine groups of body parts.

Group 1. Extremities Regular.
- Current..... 20 ma.
- Time..... 6 sec.
- Distance..... 22 in.
- Grid..... Not used
- Material..... Film
- Voltage..... Use Chart III or I

Group 2. Extremities Rapid Technique
- Current..... 100 ma.
- Time..... ¼ sec.
- Distance..... 22 in.
- Grid..... Not used
- Material..... Film with double screens
- Voltage..... Use Chart III

Group 3. Skull, Trunk bones, Pelvic Bones.
- Current..... 10 ma.
- Time..... 10 sec.
- Distance..... 30 in.
- Grid..... Used
- Material..... Film with double screens
- Voltage..... Use Chart III or I

(Skull, trunk, pelvis, rapid technique; use group 5 settings)

Group 4. Skull (Special Technique for Head Grid)

a. Anteroposterior and posteroanterior positions
- Current..... 30 ma.
- Time..... 4 sec.
- Distance..... 25 in.
- Grid..... Special head grid used
- Material..... Film with double screens

b. Lateral
- Current..... 20 ma.
- Time..... 4 sec.
- Distance..... 25 in.
- Grid..... Special head grid used
- Material..... Film with double screens

For Voltage use Chart III

Group 5. Kidney, Gall-bladder, Pregnant Uterus, Colon.
- Current..... 100 ma.
- Time..... 1¾ sec.
- Distance..... 30 in.
- Grid..... Used
- Material..... Film with double screen

Group 6. Chest Regular
- Current..... 100 ma.
- Time..... 1¼ sec.
- Distance..... 72 in.
- Grid..... Not used
- Material..... Film with double screens

Group 7. Chest Special (Use voltage as measured for anteroposterior chest)

a. Oblique Same factors except distance 50 in.

b. Lateral Same factors except distance 35 in.

c. Patient on back in bed
- Current..... 100 ma.
- Time..... 1/10 sec.
- Distance..... 30 in.
- Grid..... Not used
- Material..... Film with double screens

Group 8. Stomach
- Current..... 100 ma.
- Time..... ½ sec.
- Distance..... 30 in.
- Grid..... Not used
- Material..... Film with double screens

For Voltage use Chart III or II

Group 9. Spine and Pelvic (Low Voltage Technique)
- Chart IV
- Current..... 10 ma.
- Time..... 45 sec.
- Distance..... 30 in.
- Grid..... Used
- Material..... Film with double screens
- Voltage..... Use Chart IV

Note: One millimeter of aluminum used as a filter. "Grid" refers to the so-called Potter-Bucky diaphragm. "Film" indicates Eastman contrast film in cardboard holders. "Film with double screens" indicates Eastman film in bakelite front cassettes between Patterson intensifying screens. Using other makes of film or screen it may be necessary to alter exposure time to compensate for different speed factor.

machine but may be converted to pre-reading voltage or kilovoltage for a particular machine. By allowing for the voltage drop which occurs with 100 ma. loads, we have been able to amalgamate several charts into one. This is illustrated in Figure 3 where the horizontal values are no-load readings on the kilovoltmeter of our particular machine.

Further simplification can be achieved by marking the no-load kilovoltage scale directly on the ruler used to measure the patient's thickness. In taking a roentgenogram the technician turns to Table I for the current, time and distance factors common to the group to which the part belongs, and then by measuring with the ruler finds the kilovoltage that should be used for a part of this particular thickness.

Figure 5 is a photograph of our measuring ruler and Figure 6 is a sketch showing how it is calibrated.

This form of more or less mechanical technique is especially useful in a department where one is dealing with personnel under training, as the general principles are easy to understand and follow, and reasonably consistent results are obtained by applying the voltage scale absolutely mechanically throughout the entire range. As technicians become really experienced they develop an ability to estimate their patients as tending to be, from a roentgenological point of view, thinner or thicker than they actually measure. They learn that the stout woman with large breasts and small thoracic cavity has a chest which is denser that it measures, while the emaciated man with the large roomy thorax and thin chest wall is actually less dense. In such cases they compensate by measuring either generously or closely. They learn, too, that extremities, long immobilized by disease are much less dense than normal extremities of the same measured thickness and that the mandible requires less radiation than the lateral skull though the two may measure about the same. But all this belongs to the finesse of roentgenography.

SUMMARY

Control of roentgen exposure using kilovoltage as the sole variant is both possible and successful.

Measurement of part thickness gives more consistent results than any form of personal estimation of thickness.

It is possible to arrange all commonly roentgenographed parts of the body into nine groups within which all factors except voltage remain fixed, and then determine voltage for the particular part being roentgenographed by charting against the measured thickness of the part.

By suitable maneuvering of technique it is possible to amalgamate charts for eight of the groups into one; convert actual peak kilovoltage into no-load kilovolt readings and put these values directly on the ruler used to measure thickness of part.

The use of such a ruler is discussed.

INTERNATIONAL ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

MISCELLANEOUS

Hueper, William C. The relation of the histological structure to the prognosis of the carcinomata of the uterine cervix. *Surg., Gynec. & Obst.*, 1928, xlvii, 502–511.

A report is given of a method of numerical evaluation of histological malignancy freed to a large extent from the influence of individual interpretation by embodying twenty different factors representing histological qualities of the parenchyma and the stroma of carcinomas. The sum of the evaluations, or the "histological malignancy index" possesses a definite relationship to the end-result. The "stroma index" or the sum of the evaluations pertaining to the antiblastic qualities of the stroma was in general higher in cases with end-result 1 than in those with end-result 2, 3 and 4, dying in the first three years after the onset (end-result 1 is when the patient is free of symptoms and shows anatomical healing three years after the beginning of treatment; end-result 2, when the patient lived for two or three years after the beginning of treatment; end-result 3, when the patient lived one or two years, and end-result 4 when the patient lived a year after treatment was started).

The "parenchyma stroma" coefficient showed a definite relation to the duration of the disease. Definite information concerning the course of a carcinoma depends on numerous local and general endogenous and exogenous factors. The "histological malignancy index" in which only the histological evidence of the malignancy of the tumor cells and the local reaction of the organism is considered can therefore render an approximate estimation of the future course of the disease.

Radiosensitiveness of a carcinoma decreases with an increase of the malignancy index. A malignancy index which remains stationary or shows only minor variations from the value obtained before the beginning of the roentgen-ray treatment points to the presence of a radiorefractory carcinoma, while a marked drop of the value of the malignancy index after irradiation is apparently indicative of a good radiosensitiveness of the tumor.

Knox, Leila C. Radiosensitivity and tumor morphology. *Radiology*, 1928, xi, 229–238.

Radiosensitivity is not to be confused with clinical cure, but is a term used only to designate the action of the tissue to radiation. The attempts which are being made to correlate the morphological picture of a tumor with its radiosensitivity are of the utmost importance and should be extended as rapidly as possible. Of the morphological characteristics, the degree of differentiation is probably the most important single feature in every situation. In the use of such morphological criteria as differentiation, it must be remembered that what is true of one organ does not necessarily apply to another.

Other factors, the importance of which depends upon local conditions, are the size of the cells, size and irregularity of nuclei and the abundance of mitoses. Of less importance, is the character of the stroma and its infiltrating cells. There is no constant relationship between vascularity and sensitivity. Sensitivity may be to a certain extent predicted from morphology of a tumor, but absolute exceptions to this rule are not infrequent. In treatment, morphology, though valuable, may be subordinate to histogenesis.

Küstner, Hans. The dosage of Bucky's borderline rays in r-units determined with the standardizing apparatus. *Strahlentherapie*, 1927, xxvii, 124–145.

The use of Bucky's very soft roentgen rays has increased greatly in practice but so far it has been very difficult to get an accurate dosage. The author in this article works out the dosage of the borderline rays in r-units. The spectrum of the borderline rays with different kilovolt numbers, different thicknesses of the Lindemann glass window and different focus distances is discussed. With the same kilovolt and milliampere figures the intensity and hardness of the borderline rays proved different for different tubes because of different thicknesses of the Lindemann glass. The borderline rays are so soft that their intensity and hardness are affected merely by air absorption. For this reason the dosage of these rays must be determined at the time they are being applied. The necessity for measuring the borderline rays in r-units, as independently as possible of their hardness, because of their great absorbability led to the construction of a new kind of chamber, the short chamber. This short chamber is described in detail and illustrated. The window through which the rays pass in consists of cellophan treated

with graphite; it is a few hundredths of a millimeter in thickness and the physician can change it himself when necessary. This chamber can be placed in the standardizing apparatus. The absorption of the borderline rays in air, cellon and cellophan was determined. It was found that the absorption in 10 cm. air was equal to that in 0.105 mm. cellon. The short chamber was compared between 7 and 60 kv. with the small containing chamber and between 20 and 60 kv. with the containing chamber of the large standardizing chamber and the short chamber standardized in R-units.

The deep distribution of the borderline rays in cellon was determined and that in water calculated from known data. The intensity down to several millimeters depth is by no means negligible. The depth of penetration of the borderline rays differs so much with different kilovolt counts that it must be determined too. This is best accomplished by an analysis of absorption. The dosage with a dermix apparatus showed that with 10 cm. focus distance, 9 kv., and 9 ma., 1000 R-units were given in 7½ minutes.

KÜSTNER, HANS. The absolute determination of the R unit with the large gauging apparatus. *Strablentherapie*, 1927, xxvii, 331–347.

A method is described and illustrated which makes it possible to determine the R-unit absolutely with the author's large gauging apparatus. A Harms condenser of known capacity is attached to the apparatus and its middle coat connected alternately with the covering of the apparatus and with a known source of current in such a way that on applying the current to the electrometer cords of the gauging apparatus it runs through the same scale as in ionization by roentgen and radium rays. The amount of electricity produced in this way in the gauging apparatus by way of ionization is then independent of the capacity and current sensitiveness of the gauging apparatus as well as of the capacity of the connecting conductor and equal to the product of the capacity of the Harms condenser and the current. A gauging obtained in this way by connection with the condenser also holds good after detachment from the gauging apparatus. In this way the absolute measurement can be transferred to instruments (condenser, voltmeter) which can be gauged, controlled and kept independent of the gauging apparatus. Two measurements taken independently of each other show that the gauging of gauging apparatus by the absolute method agrees to within 1 to 2 per cent with gauging by comparison with a normal apparatus.

LAHM, W. Reporting dosage in radium treatment. *Strablentherapie*, 1927, xxvi, 773–780.

This is a criticism of Coliez' suggestion for reporting dosage in radium treatment. The author concludes that there is no reason for giving up the usual designation of amount and dosage based on the content of the preparation in radium element. Different conceptions only introduce uncertainties, which would better be avoided, into the difficult question of the use and dosage of radium. So it is best to continue the use of the milligram-hour as the unit of dosage, particularly if it is supplemented by the conception of local radium element concentration and the determination of the distance in centimeters. In the special technique of irradiation it will always be necessary to state accurately the amount of radium, the form of the carrier, the filtration, distance and interval.

MIESCHER, G. Roentgen biology of the normal and diseased skin, *Strablentherapie*, 1927, xxvii, 257–280.

On the normal skin the clinical symptoms of the roentgen reaction are redness, pigmentation, loss of hair and in severer degrees erosion and formation of a scab. The reaction was formerly thought to take place in two waves, an early erythema immediately after the irradiation and the true roentgen erythema at the end of the second week or in the course of the third. But it has been found that it takes place in three or sometimes four waves, during the first, third, sixth and ninth weeks. Sometimes they are separated by intervals, sometimes they become confluent. The hardness of the rays has no effect on the course of the reaction. The French authors are mistaken in saying that there is an elective injury of the epidermis by hard rays. And recent experiments made by the author have shown that the child's skin is not more sensitive than that of the adult. It even seems to be a little less sensitive. The skin erythema is always followed by pigmentation. In weak reactions pigmentation may even occur without any visible redness. Signs of degeneration occur early in the cells of the epidermis. The most interesting histological change is the anomalies of mitosis. From the moment of irradiation until regeneration begins there are no normal mitoses. When regeneration begins, after several weeks, many normal mitoses suddenly appear. The time when regeneration begins does not seem to depend on the dose at all but only on the degree of injury of the skin. Cells that have been very little injured or not at all are the starting point of the regeneration. They seem

to be reserve cells that are not sensitive to rays and are found in other tissues also.

Functional and capillary microscopic examinations show that the waves produce inflammation rather than injury of the vessels. There is no proof that immunobiological processes are involved in the roentgen reaction. No actinoproteins are known that act as antigens and there is no evidence of the development of antibodies. The biological nature of the roentgen skin reaction is still by no means clear. When the acute dermatitis is over with there is a late injury characterized by telangiectasis, anomalies of pigmentation and necroses and sometimes ulceration and the development of carcinoma. In roentgen ulcer there is no granulation tissue demarcating the necrosis. In the late injury as well as in the acute dermatitis the capacity of the cells for normal mitosis is destroyed, but in acute dermatitis the greatest change is in the epidermis, in late atrophy in the cutis. The degree of cumulation of the roentgen effect varies in different cells and tissues.

In pathological conditions roentgen irradiation is valuable in abscesses, furuncles and phlegmons. Pain and swelling quickly disappear and the course of recovery is shortened. This may be due to an action on the leucocytes or an action on the general condition analogous to the action of protein bodies. In late eczema, in which the effect of the rays is also good, they probably destroy the cells that are most injured so the skin has a chance to recover. No systematic histological examinations have been made of the action of the rays in eczema. There is no decrease of the allergic reactive capacity of the skin brought about by roentgen rays. After healing of an irradiated as well as an unirradiated focus of eczema the skin remains as specifically hypersensitive as before; the same is true of other forms of allergy, for example tuberculin allergy. Roentgen rays do not have a desensitizing action.

Motojima, R. Experimental study of the effect of roentgen rays on inflammations in comparison with other physical methods. *Strahlentherapie*, 1928, xxix, 30–70.

Experiments are described on rabbits. Catgut threads impregnated with silver nitrate or silk threads impregnated with emulsions of streptococci were run beneath the skin of the animals, producing inflammation; they were then irradiated with 180 kv., 23 cm. skin distance, 0.5 mm. copper filter, one skin erythema dose, or 720 r. The experiments confirmed the fact, found clinically, that roentgen rays have a good effect on inflammations. The inflammatory infiltration around the source of irritation is decreased. There is a certain breadth of dosage that is effective which varies with the species of animal and the local condition of the tissues. Local and general processes play a part in the effect. The general processes are ones of nonspecific increase of immunity; the local ones consist primarily of dilatation of the vessels, hyperemia, increased movement of lymph and processes of local immunity. Formation of abscesses can be furthered by large dosage. The effect of roentgen rays in inflammation resembles that of heat most and that of cold least. Combined heat and roentgen treatment with proper dosage is more effective than either one alone.

Murdoch, J., and Stahel, E. Comparative studies of two dosimetric roentgen units—the French r (Solomon) and the German r (Behnken). *Strahlentherapie*, 1927, xxvii, 561–571.

Comparisons made by the authors show that the relation between the two r units is not constant. It varies from 2.40 to 4.55 in the measurements that they made. The proportion increases with an increase of tension and filtration. Ionometric chambers of different construction which were gauged with radium according to Solomon's directions very rigorously in the same way gave very different figures. The French unit therefore cannot be considered an absolute unit. It is essential that the small ionization chambers in ordinary use be gauged with the aid of large standard chambers for the different tensions and the different filters used. The ionometric measurements must be made in the air without a phantom if an ionization chamber is not available whose figures are independent of wave lengths. But it is possible, taking into consideration the secondary irradiation measured by Solomon's apparatus, to compare the doses of French and German authors. If these different factors are taken into account it seems that the disagreement in the two units is not very great.

Nasledow, D. N., and Kacura, T. M. Dependence of distribution of deep dosage on the kind of roentgen apparatus. *Strahlentherapie*, 1927, xxvii, 169–186.

The question of whether the distribution of the deep dosage depends on the kind of apparatus used is of fundamental importance in the question of roentgen dosimetry. In this article the author compares the distribution of dosage in the deep tissues with different forms of

apparatus and gives curves showing the results. The measurements were made with a Siemens dosimeter. They showed about the same values for percentage dosage in the deep tissues and certain differences in absolute values; these differences decreased in therapeutic apparatus with increasing tension and heavy filters. The quality of radiation yielded by three different types of apparatus was also examined. It was found that a mixed apparatus resembled the stabilovolt apparatus closely in the quality of rays yielded while the hard ray apparatus differed considerably from both. The radiation yielded by the stabilovolt apparatus was 1.5 times as intense as that of the hard ray apparatus. These results were obtained by a study of the microphotograms of spectra (Koch's microphotometer) and the half-value layer.

PAULI, W. E., and DENNIG, H. The permeability of animal and human skin in the visible part of the spectrum. *Strablentherapie*, 1927, xxvi, 761–766.

This article describes the measurement of the permeability of the skin in the visible part of the spectrum and gives a curve showing the total course of permeability for the rays of the spectrum from about 450$\mu\mu$ to 1000$\mu\mu$.

PAULI, W. E., and KLIEWE, H. Artificial increase of the permeability of the skin to bactericidal light rays. *Strablentherapie*, 1927, xxvi, 767–772.

In a previous number of *Strablentherapie* the authors reported a method of artificially increasing the permeability of the skin to short wave rays of light. This increased permeability was demonstrated by blackening photographic films. In addition to making it possible for short wave rays to penetrate the tissues deeper it seemed that this method would make it easier to kill or inhibit the growth of bacteria beneath the skin by the use of the short wave rays. If so it would be useful in phlegmon, panaris, etc. In this article they describe experiments on this point. They irradiated staphylococci and streptococci through normal skin and through skin whose permeability had been increased by the above method. They found marked inhibition of growth of both kinds of bacteria after twenty minutes' irradiation through prepared skin while there was no inhibition of growth through normal skin.

PAULI, W. E., and SULGER, E. The bactericidal action of roentgen rays and its dependence on external factors. *Strablentherapie*, 1928, xxix, 128–138.

The authors' experiments in irradiating cultures of different kinds of bacteria showed that the bactericidal force of roentgen rays is dependent to a great extent on temperature. This is probably due to the fact that an increase of temperature increases the rapidity of division of the microorganisms, and it is known that cells are particularly sensitive to roentgen rays during karyokinesis. The same thing is true of plant embryos, and that is the reason why sex cells which are undergoing rapid division are so much more sensitive to roentgen rays than nerve cells, for instance.

They also found that giving the irradiation in fractional doses, one immediately after the other without an interval between them greatly increased the bactericidal force of the rays. This is because during a long-continued irradiation every cell at some time reaches this stage of roentgen sensitiveness. The more the irradiation is divided, the greater the probability that it will injure all the cells of the culture. The arguments that Holzknecht and his school used against irradiating tumors with a large dose all at once hold good in the irradiation of bacteria as well.

They also found that colloids by their secondary irradiation increase the bactericidal action of the rays, a fact that had previously been found by other authors in a different kind of experiment.

PETER, GUSTAV. The question of vessel permeability in the irradiation of malignant tumors. *Strablentherapie*, 1927, xxvi, 735–748.

The author was obliged to adopt the method of repeated small doses because he found that on the high plateau of Mexico (2300 meters above the sea) it was impossible to use a single large dose because it caused roentgen sickness and particularly injured the blood, without doubt due to the lack of oxygen. This in connection with a clinical case of epithelioma of the scleral part of the conjunctiva which is described led him to believe that the curative action of the rays is due to a change in the vessel wall and this must be a change in its permeability for certain substances. The possibility that vessel permeability is important both in the origin and cure of cancer is also indicated by other clinical facts, such for instance as that paralysis, syphilitic ulcers of the leg and cancer may heal after intercurrent erysipelas or scarlet fever. The same thing is seen in keloids, eczema, psoriasis and lupus, that is, diseases that can be cured by roentgen rays. It seems probable that the erythema in these cases is responsible for the cure. And in connection with the immunity conditions in

syphilis it seems probable that the erythema dose which cures cancer increases vessel permeability for certain substances. It also seems probable from certain experiments that are described that normal oxidation is the protective function which prevents the origin and development of cancer. The value of irradiation then would lie in increasing permeability to oxygen itself or to oxidative ferments and other catalyzers. Therefore the objects of therapeutic technic should be to give a dosage of rays which would lead to long-continued hyperemia and increase of permeability without injuring the vessels so that they could not react to later irradiations if necessary, and to combine roentgen therapy with the administration of substances that further oxidation at the expense of fermentation and prevent hyperacidity of the tissues. The author gives alkalies until the reaction of the urine is alkaline, and thyroidin and iron. The best dosage seems to be that recommended by Anschütz and Hans Meyer in Kiel for carcinoma of the breast, that is ½ to ¾ H.E.D. eight times a year. It will require further experimental investigation to show the minimum dose that meets the requirements for increase of permeability.

Politzer, G. The specific action of roentgen rays. *Strahlentherapie*, 1927, xxvii, 533–544.

By specific is meant a kind of action peculiar to the sort of stimulus used and not exercised by any other kind of stimulus. The changes in mitoses in irradiated tissue are examples of the specific effect of roentgen rays. There is a primary effect, an interval free of mitoses, and a secondary effect. The primary effect is characterized by pseudo-amitoses and pycnoses; the secondary effect by destruction and deviation of the chromosomes and formation of partial nuclei. But after having shown these effects it is necessary to show that they are not produced by any other kind of stimulus. The author made experiments with vital stains which color the cells without injuring them and found that they caused changes similar in many ways to those produced by roentgen rays, but not the characteristic secondary effect. Experiments by other authors are also described which show that electromagnetic waves of different wave lengths not only have different effects but sometimes directly opposite ones. So experimental work seems to show that roentgen rays do have a definitely specific action.

Pugno-Vanoni, E. Roentgen treatment with small diagnostic apparatus. *Radiol. med.*, Dec., 1928, xv, 1213–1215.

An illustration is given showing how a simple apparatus for diagnosis without a rectifier can be transformed by the addition of two condensers and a valve so that it can be used for a therapeutic apparatus that is more than sufficient for many uses. It can be used for tensions up to 190 kv. and currents up to 6 ma. without losing its value for diagnostic purposes. The type of ray produced is sufficient even for deep therapy if only a limited number of treatments are to be given.

Report of the Committee on Standardization of *x*-ray measurements of the Radiological Society of North America. *Radiology*, April, 1928, x, 318.

"1. The unit of effective *x*-ray intensity, "*one* R," is that intensity of radiation which produces a saturated ionization current of one electrostatic unit per cubic centimeter of a non-restricted volume of air at a temperature of 0° Centigrade and a pressure of 760 mm. of Hg.

"2. The method recommended by the Committee for the measurement of *x*-radiation in terms of the above unit in a nonrestricted volume of air is the employment of an air ionization chamber—

(*a*) with open windows at the sides of incidence and energence;

(*b*) with guard electrodes sufficiently large to insure the desired electric field;

(*c*) with suitable spacing of electrodes to include sensibly all of the ionizing effects of the photo-electrons;

(*d*) an arrangement of diaphragms to properly collimate the beam and prevent undesired secondary radiation effects.

Such an ionization chamber constitutes an "absolute standard."

"3. The measurement of the *x*-radiation at the place of practical application may be made by a dosimeter which is calibrated in R-units by means of an "absolute standard." Such a dosimeter constitutes an "absolute standard."

Preferably a practical dosimeter should have an ionization chamber in which the measured ionization current is proportional to that produced in an "absolute standard" with the various qualities of radiation employed. The constancy of such a dosimeter may be satisfactorily controlled by means of a suitable radio-active substance properly employed.

"4. Voltage and filtration alone do not properly define radiation quality. Spectrometric analysis of the *x*-radiation gives the most complete determination of its quality. For practical purposes the quality may be expressed by the half value layer in a suitable

material (copper or aluminum is recommended) or by the effective wave length.

The half value layer for the *x*-radiation employed is that thickness of the given material which will reduce the reading of the "absolute standard" or "calibrated standard" to one-half of its value.

The effective wave length of the *x*-radiation employed is the wave length of monochromatic radiation for which the readings of the "absolute standard," or "calibrated standard," would be reduced in the same ratio as that actually observed when a certain thickness of a given material (copper or aluminum) is interposed.

"5. The Committee recommends that dosage be expressed in terms of R-units and time of application, and that the quality of the radiation be stated in terms of effective wave length or half value layer.

Respectfully submitted,
Committee on Standardization of *X*-ray Measurements
EDWIN C. ERNST, M.D.,
Chairman.
OTTO GLASSER, PH.D.,
Sub-chairman."

ROST, F., and DIETERICH, W. The effect of roentgen rays on vitamine-free food. *Strablentherapie*, 1928, xxix, 152–160.

Experiments are reported in which rats were fed with non-irradiated food and with the same food after irradiation with roentgen rays. Adult rats were used for the experiments and the food was deprived of vitamines by heating before it was irradiated. With this method of experiment the food was not injured by roentgen irradiation. On the contrary, when rats had been fed with vitamine-free bacon that had not been irradiated they seemed to do somewhat better when they were given bacon that had been irradiated. But the improvement was not so marked as when they were fed with food given the same preliminary treatment and then irradiated with ultraviolet rays.

ROSTOCK, PAUL. Unusual pigmentation of the hair after roentgen irradiation. *Strablentherapie*, 1928, xxvii, 800–802.

Breitländer and Felke recently described a case of dark pigmentation of the hair after irradiation in a boy aged fifteen who had blond hair. The author has recently seen a similar case in a man of thirty-two in whom the neurological findings indicated a tumor in the left hemisphere. On May 18, 1925, a trephine was performed but nothing pathological was found so the wound was closed. The patient was then given roentgen irradiation on left, right and posterior fields which resulted in improvement in the neurological findings. Hard rays were used with a filtration of 3 mm. aluminum and 0.5 mm. zinc. After the first irradiation there was a complete epilation of all the fields. The patient had been almost bald but when he came back for another irradiation on Feb. 12, 1926, there was a normal growth of hair on the right side and on the occiput, but the hair on the left side had become very much darker than the rest of the hair which was blond. It was much coarser than the normal hair and the cortex was intensely pigmented while the center contained air. In this case the dark hair was found only on the site of operation and not on the other fields of irradiation but the operation could not have been the cause of it as no operation had been performed in Breitländer and Felke's case. The cause of the phenomenon is not known.

ROTHER, JULIUS. Experimental study of the effect of roentgen rays on sugar. *Srtablentherapie*, 1927, xxvii, 197–256.

Irradiation of the upper part of the abdomen of dogs, guinea pigs and rabbits with a large dose of roentgen rays caused a rapid rise in blood sugar values. In one to one and a half hours sugar rose from a normal of 0.10 per cent to about 0.17 to 0.19 per cent, and then fell gradually. If the irradiation was given after two days' fasting the rise of blood sugar did not take place. It was found that the sugar came from the liver whose glycogen supply was decreased after the irradiation. Isolated surviving liver tissue was then examined to see whether the glycogen supply would be decreased after irradiation. But even intense irradiation of either suspensions of liver tissue or of the liver removed whole from the body did not show any effect on sugar or amino acid nitrogen.

The examinations also showed that the increase in blood sugar does not take place after roentgen irradiation, or is even changed into a fatal fall of blood sugar if the splanchnics are sectioned on both sides. An injection of ergotamin, which is a vegetative toxin that has an inhibiting action on the sympathetic, has the same effect as splanchnicotomy. This shows that the vegetative connections of the liver with the organism must be intact in order for a hyperglycemia to take place on irradiation.

Liver suspensions of guinea pigs irradiated during life in addition to the glycogen poverty mentioned above showed a decrease of sugar and of post-mortem production, as well as an increase in the amino acid nitrogen content and post-mortem amino acid production for an hour in the thermostat. There were no histological findings to account for this functional impairment. With the doses used death occurred in five days. The functional disturbance was greater the longer the time between the irradiation and death. The injury was the same whether the immediate liver region or the lower part of the abdomen was irradiated. On autopsy the gastrointestinal tract of the animals was found full, so hunger could not have been the cause of the death of the animals. It is more probable that injury of the intestines by the rays caused them to give off toxic products into the circulation.

There are two possible explanations for the effect of irradiation: first, that it is due to injury by products of disintegration of cells affected by the rays, and second that it is due to the action of the rays on colloid electrolytes. The author thinks the latter is the more plausible.

Rottmann, H. G. Histology of borderline ray erythema. *Strablentberapie*, 1927, xxvii, 518–532.

Changes demonstrable microscopically can be found in the skin of the subjects a few hours after the irradiation when borderline erythema is barely beginning macroscopically. They increase considerably in the course of the next two days, have not decreased in strength after eight days, are weaker after twenty-five days but still visible after three months. The action of the rays can be seen in all the layers of the epidermis and in the vessels of the corium and on overdosage also in the follicles, without involvement of the parenchyma of the sebaceous glands, and in the upper part of the connective tissue (edema). The sweat glands generally do not show any structural changes; slight swelling of the cells and nuclei was visible only with the largest doses. The changes in the epidermis consist of edema of the cells and nuclei, vacuolization and contraction of the nuclei. There were granular changes in the stratum granulosum which on giving three erythema doses could also be seen in spots in the upper cell layers. There were always vessel changes in the corium: swelling of the endothelium, great changes in the adventitia cells, perivascular cell infiltration, chiefly lymphocytes, fewer leucocytes. These changes are not limited to the upper capillaries but also extend to the deeper vessels, about to the fundus of the sebaceous glands. On the use of large doses there is infiltration around the sebaceous glands and hair follicles. The retrogression of the anatomical boundary-ray erythema is very slow. For three months after the irradiation there are still changes in the superficial vessels of the corium and an intense pigment deposit in the basal cells. A histological comparison between boundary-ray and roentgen-ray erythema shows that they are strikingly alike qualitatively. Aside from the changes in the stratum granulosum that are not seen in roentgen erythema the two can hardly be differentiated.

Rubin, I. C. Observations on the intramural and isthmic portion of the fallopian tubes with special reference to so-called "isthmospasm." *Surg., Gynec. & Obst.*, January, 1928, xlvi, 87–94.

The intramural portion of the fallopian tube in the living pursues a straight course, contrary to what is found in anatomical specimens whether fresh or formalinized. It is capable, like the rest of the tube, of contracting and dilating. Total obliteration of its lumen is very infrequent and when present is part of more extensive tubal disease. "Isthmospasm" as an entity has not been definitely demonstrated, but tubal contractions can be seen with the aid of intrauterine lipiodol injection and the roentgenoscope. The uterotubal junction (intramural portion) appears to be the site of predilection of spasm or hypertonicity in a relatively small proportion of the cases examined. It was demonstrated by uterotubal insufflation with the aid of the kymograph in 2 per cent of 450 cases of sterility. It can also be demonstrated by roentgenoscope and roentgenogram aided by lipiodol and manometric control. A sphincteric action at the tubo-uterine junction has been evidenced in clinical and experimental observations.

From roentgenological studies the tube may be divided into two main portions; the first a narrow proximal, uterine portion, and the second a wider distal abdominal portion. The length of the fallopian tube, as revealed by salpingography, is greater than is apparent on inspecting it at the operating table or in anatomical examinations. In the living state it frequently measures 6 or 9 inches as against 3 or 4 inches when removed from the body. This extended length of the tubes in the roentgen films may be due to a straightening out of the tube lumen through the force of the injected

fluid. Magnification of the picture on the film is very slight.

SAUER, FRITZ, and OBERGUGGENBERGER, VIKTOR. Roentgen rays and electrical conductibility. *Strahlentherapie*, 1928, xxviii, 589–597.

Results of the authors' own experiments and of those reported in the literature on the influence of roentgen irradiation on the electrical conductibility of the irradiated substance show that the rays bring about a great change in gases and non-conductors and very little in metals and electrolyte solutions. A method based purely on the principle of energy and illustrated by two examples gives results that agree very well with those of the experiments so they may be considered sufficiently reliable.

SCHMITZ, W. Roentgen energy measurements with the selenium intensimeter. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1927, xxxv, 684–697.

A careful series of measurements were made upon a Siemens diagnostic apparatus and a radiosilex apparatus using the following tubes: (1) a Müller water-cooled copper tube for measuring the copper Kα line; (2) a Müller water-cooled material investigating tube; (3) a Lilienfeld molybdenum tube, and (4) a radiology therapy tube. Measurements were made of the wave length and of the intensity in R-units. Following this, measurements were made of the absolute values of the intensity in terms of ergs per square centimeter using the air thermometer and bolometer. Measurements were also made of the effect of the rays defined as above upon the selenium cells in terms of "F" units with consideration of resistance in ohms and constancy or fatigue of the cells.

An analysis of the results shows that the following points must be considered in the practical application of the selenium intensimeter: (1) It is desirable to have the readings of the instrument greater than 1 F in order to overcome the inertia of the apparatus. This requirement is not easily fulfilled when measuring hard rays. (2) If the selenium intensimeter is to be compared with ionization instruments, the wave length of the radiation should be known. It is desirable to calibrate the intensimeter in terms of energy values. The calibration can be checked with any wave length and the procedure is valid for the entire range of wave lengths. (3) The intensimeters studied exhibited satisfactory constancy in their readings.

SCHNEIDER, G. H. Important technical points in establishing a new roentgen institute for treatment. *Strahlentherapie*, 1927, xxvi, 792–798.

In establishing a roentgen institute it is not advisable to install a universal apparatus for diagnosis and treatment both, as it will be disappointing when great demands are made on it for diagnosis and particularly for treatment. And besides in case of repair it will be necessary to stop both the work of diagnosis and of treatment. In choosing apparatus for treatment it must be remembered that an apparatus of the stabilivolt type, the Veifa for instance, can also be utilized as an auxiliary in diagnosis, though of course its irradiation is not as well adapted for that purpose as that of an apparatus especially constructed for diagnostic purposes. A further increase of tension and current strength over that of the types of apparatus heretofore in use is not only useless but unfounded both medically and biologically and necessarily involves great danger; and care must be given to the conduction of the current in order to ensure uniform working conditions and guarantee a certain dosage in a certain time. The cross section of the wire that carries the current from the source is of great importance. The current should be derived from near the source before lines for lighting, etc., have been given off as that will ensure greater uniformity and strength of current and inside the hospital the same wire should not be used to supply electric chairs and other apparatus.

SCHNEIDER, G. H. The importance of exact anatomical measurements in roentgen therapy. *Strahlentherapie*, 1928, xxvii, 751–756.

The author describes a combination compass for all purposes of roentgen measurement from a body cavity or a point on the surface of the body to another point on the surface of the body which is to be used as the point of incidence for the central ray in a field for roentgen irradiation. At the same time the external roentgen compass can be used for an obstetrical compass. The way of taking the measurements and their purpose is described, and the use of the method in irradiation of the prostate illustrated. The importance of external measurements in obstetrics is compared with it and emphasized. The external conjugate measurement of the pelvis, Baudelocque's diameter is compared with a roentgen Baudelocque which can be used for irradiations of the pelvis in both man and woman. It is the distance from the plane of the anterior border of the symphysis to that of the posterior wall of the sacrum.

SCHREUS, H. Th. Gauging the Sabouraud-Noiré dosimeter in R units and its usefulness in practical dosimetery. *Strablentberapie*, 1928, xxix, 375–384.

Critical tests of the Sabouraud-Noiré dosimeter with the physical and biological methods in current use for gauging in R units showed that it is quite inaccurate in some respects, which is hard to reconcile with the results of years of practical use of the method. The results shown by the tablets are dependent on wave length. But with careful use and sufficient practice the dosage can be determined within 15 to 20 per cent and its dependence on wave length can also be compensated by taking into consideration the hardness of the rays. The tests show very high R values for the erythema dose and therefore the dosimeter should be tested further and the erythema dose decreased if necessary.

SCHUGT, P. The effect of graduated doses of roentgen rays of different wave lengths on the structure and function of the ovaries. *Strablentberapie*, 1928, xxvii, 603–662.

Experiments on white mice are described and histological pictures given. The castration dose for the white mouse was found to be 54 R of a hard radiation. The same effect was obtained with a smaller dose, 42 R, of a soft radiation. Down to doses of 21 R there was an injury of the follicles consisting of a decrease in the number of follicles. The primordial follicles were not an exception; wherever there was injury of the other follicles the primordial follicles were injured also. These results show that temporary castration is not absolutely harmless; they also show that the theory that the primordial follicles are most resistant and are preserved even when the others are destroyed is at least doubtful. There was no evidence of a stimulant action of the rays in this material. Up to seven months after administration of a castration dose or more up to 140 R there were remnants of follicular epithelium and corpus luteum tissue but no ova. Changes were found in the interstitial tissue of the ovaries of the castrated animals like those that have frequently been described as characteristic of the interstitial gland. No differences could be demonstrated in the function of the ovaries irradiated with hard and soft rays. Rut did not stop after the administration of the castration dose or more up to 140 R. It was demonstrated after four and a half months in all the 45 animals examined and after seven months in 11 of 19 animals examined.

Roentgen rays destroy the external secretion of the ovary which leads to ovulation but not the internal secretion of the hormone that causes rut. The production of the rutting hormone in the ovaries could not be stopped with doses up to two and a half times the castration dose and up to a time six months after the irradiation.

SCHUGT, R. Experimental investigation of injury of offspring by roentgen rays. *Strablentberapie*, 1928, xxviii, 546–567.

Experiments in irradiating mice and pairing them afterwards are described and the details of the results shown in tables. There were no malformations of any kind in the offspring but those of irradiated mothers did not develop so well as those of non-irradiated ones, and the fertility of the irradiated mothers was decreased. The control mice became pregnant in 100 per cent of the cases while only 61.9 per cent of the irradiated females paired with normal males ten days after the irradiation became pregnant; of those paired three and a half months later only a few that had been given less than the castration dose became pregnant. When paired with each other the offspring of the irradiated mothers remained sterile. When the females were paired with normal untreated males 40 per cent of them became pregnant. This indicates that the male offspring are injured more than the females. And the average number of young produced was somewhat less than for normal animals. The estrual cycle of offspring of irradiated mothers was not strikingly abnormal. Many animals showed rut and did not become pregnant while in others the cycle was quite irregular and yet conception took place. More or less regular rut therefore does not exclude sterility.

The author's results do not support the theory that the malformations reported by some authors are really due to injury from the rays in irradiation of the uterus. The only injury in the author's experiments was underdevelopment and decreased fertility.

SCHULTZE, G. K. F. Dosage in irradiation of carcinoma of the uterus in R units determined by vaginal measurement. *Strablentberapie*, 1928, xxviii, 524–545.

The skin erythema dose is not a reliable method of measurement so a dosage in R units should be substituted for it. The author describes his method of determining surface dosage by means of a water or paraffin phantom, gives illustrations showing the fields and the mathematical calculations by which the distribution of intensity is determined.

He then made direct measurements on the patients to see whether the doses actually given correspond to the ones calculated. His direct measurements showed that in spite of every care it is not possible to determine accurately by calculation just what dosage will be attained in the pelvis; the calculated values are only relative and must be controlled by direct measurements. These measurements were carried out by introducing the chamber of the Hammer dosimeter into the vagina during the whole period of irradiation and counting the number of electrometer discharges automatically. Care must be taken always to put the chamber in the same position. Such continuous measurements of course can only be made with an instrument with an automatic arrangement for discharge and counting. The number of electrometer discharges at the end of the irradiation need only be noted and calculated into R values from the tables. This gives the absolute measurement of the total dosage in the pelvis. The dosage used by the author for the irradiation of carcinoma lay between 600 and 800 R.

SILVERMAN, DANIEL N., and DENIS, WILLY. On the relationship of gall-bladder emptying to ingested fats. *Radiology*, July, 1928, xi, 45–47.

The authors have shown that while emulsified fats when ingested produce emptying of the functioning gall-bladder, absorption of the fat into the blood bears no relation to this process.

SIMON, MORITZ. Stray remarks on the history of medical radiology in Sweden. *Acta radiol.*, 1926, vii, 476–490.

Brief accounts are given of the activities of the first three Swedish roentgenologists, namely, Dr. Thor Stenbeck, Dr. T. Sjögren, and Dr. I. Bagge. Remarks are also included on the roentgen-ray work done in Swedish hospitals during the first decennium of roentgenology. A more detailed account is given of the daily roentgenological work in 1907 at the roentgen institute of the Seraphim Hospital. Finally there is included a tabulation of the various Swedish hospitals and sanatoria and the nature of their roentgenological work.

SOLOMON, ISER. Determination of the quality of roentgen radiation in practice. *Strahlentherapie*, 1928, xxix, 534–542.

The author believes that the quality of radiation should be measured by the absorption method. The results of this measurement should be expressed in degrees of permeability of a layer of sheet copper 0.5 mm. thick or in terms of the copper half-value layer. It is preferable to work with measuring apparatus that permits of immediate and continuous reading of the degree of permeability of the radiation used.

SPIEGLER, GOTTFRIED. A circuit for protection of the tube. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1927, xxxv, 786–787.

A description is given of a circuit which makes it impossible to apply current to a Coolidge tube without the heating circuit being turned on. The arrangement at the same time switches off the high tension current should the filament circuit fail during operation of the tube. The essential feature of the apparatus is the employment of a solenoid through which the filament current must pass. The solenoid controls an auxiliary circuit which in turn controls the opening or closing of the high tension circuit.

STEINBACH, R. Method for measuring distances in the interior of the body upon the roentgen screen, illustrated by measurements of the transverse diameter of the heart. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1927, xxxv, 1259–1264.

If the plane of the screen is placed parallel to any line whose depth in the interior of the body is known, its true length may be easily calculated from the geometry of the system. The technique followed by the author in the case of measurement of the diameter of the heart is as follows: The patient is placed against the stand of the roentgenoscope and the screen is brought close to the chest. Care is taken to have an exact frontal position. After measurement of the apparent transverse diameter, the screen is carefully moved 8 cm. away from the patient and another measurement is made. The difference between the two measurements subtracted from the length first measured gives the required transverse diameter of the heart. This is made on the assumption that the transverse diameter lies 8 cm. from the anterior surface of the thorax. A consideration of the sources of errors shows that a slightly oblique position of the diameter produces an error in length which is compensated for by the fact that the assumed 8 cm. depth is usually less than the actual. A series of 16 patients were measured by the author's roentgenoscopic method and by teleroentgenograms, with the result that the average deviation was only 3.5 per cent from that found in the teleroentgenograms.

Stoklasa, Julius. The effect of radioactivity of water on biological and biochemical processes in the cells of lower and higher organisms. *Strahlentherapie*, 1928, xxix, 324–332.

Radioactive springs are rich in oxygen. The more strongly radioactive springs contain many bacteria and algae. The development of iron bacteria is particularly furthered by radioactivity. The typical fresh-water animals develop very intensely in radioactive waters. The flora and fauna are ordinarily richest in moderately acid radioactive waters in which the hydrogen ion concentration is 6.2 to 6.8 and the minimum radioactivity 2.6 mache units or 9.464 eman, and which contain at least 7 c.c. O_2 per liter.

Water was kept in small glass reservoirs and radioactive air conducted into some of them and air that was not radioactive into others. The experiments showed that the assimilation and accumulation of elementary nitrogen is increased by radioactivity. This explains why radioactivity furthers the development of green algae.

In comparing the water of different springs it is found that the best therapeutic effects are obtained with the waters of the springs richest in radium emanation. Water containing radium emanations has a good effect in infectious or traumatic inflammations, diseases of the central nervous system, disturbances of internal secretion, disturbances of metabolism and constitutional anomalies. There is now no longer any doubt of the therapeutic effectiveness of radioactive waters. Radium emanation furthers all enzymatic processes if oxygen is present in sufficient amounts. Therefore for practical therapeutic purposes oxygen should be conducted into the radium bath, and during or after the bath the patient should inhale oxygen. The greater the amount of oxygen available, the more of the radium emanation can be utilized for enzymatic processes. The action of oxydases and peroxydases is greatly increased by radium emanation. Radium emanation has a good effect on spermatogenesis and ovulation.

It seems very probable that in many cases cells have a selective action for alpha or beta rays. This fact and the different degree of energy of the different kinds of rays probably explains some apparently paradoxical effects of irradiation with radioactive substances.

Takeda, T. The biological effects of incorporated radioactive substances (thorium x rods) from experiments on testes. *Acta radiol.*, 1927, viii, 130–145.

A study was made of the reactions occurring in the tissue of a very radiosensitive organ, the testicle, produced by the introduction of rods of thorium x. The progress of the histological changes in the tissues were followed from two hours to ninety-seven days. The initial strength of the inserted thorium x was varied in order to establish the relation of the extent of changes produced to the strength employed. The experiments were performed on mature rats using thorium x rods of 0.4 mm. diameter and 0.25 to 0.50 cm. long, containing 0.5 mc. per cm. The effective radiations emitted were of the beta and gamma type. The alpha radiation was practically screened off by inert material present. During the early stages of irradiation there occurred infiltration of leucocytes with pycnotic signs in the cells. Immediately adjacent to the radioactive rod the spermatogonia showed slight degeneration. After five days' exposure the testis was pronouncedly hyperemic. Immediately adjacent to the rod the contours of the canaliculi were only faintly visible and their interior was filled with cell shadows and debris. There was also present the debris of the leucocytic infiltration occurring during the early stage. The necrotic zone was about 2.5 mm. in diameter and at its boundary there was a dense infiltration of partly injured leucocytes, the small vessels being congested. Nowhere in the preparation were there any signs of spermatozoa. With increasing time of exposure the necrotic area increased in diameter, and there developed a dense network of connective tissue followed by pronounced contraction of the organ.

Tarantino, E. Measuring high tensions with the spintherometer and relative corrections. *Radiol. med.*, May, 1928, xv, 454–462.

Tarantino reviews the methods of measuring the high tensions of roentgen apparatus and then discusses spintherometers, and particularly the corrections which must be made in order to get good spintherometric measurements. He gives four tables which will help the roentgenologist in measuring tension by the new apparatus. The first of these tables shows the curves of the different types of spintherometer; the second shows a diagram of the factor of correction; the third shows the variations in barometric pressure due to altitude; and the fourth shows very clearly all the results given by the preceding tables in relation to normal conditions of pressure and temperature.

Taylor, Henry K. Potter-Bucky table adapted for multiple exposures. *Radiology*, June, 1928, x, 506–509.

The device described was adapted to an Acme-International Potter-Bucky table No. 3, within the depressed portion of which is placed a piece of sheet lead (4 pounds to the square inch) measuring 20 × 25 inches. In the exact center of the lead sheet an opening 5½ × 4⅔ inches is cut, the larger measurement being crosswise. The opening allows for six exposures on a 11 × 14 film. This lead sheet is enclosed in a tightly fitting black vello cloth envelope. It is not necessary to remove this from the table when doing routine work, for it is easily displaced to either end of the table. Within the Bucky tray three strips of wood are placed which permits the entry of an 11 × 14 cassette and also permits a displacement of 5½ inches crosswise. The table is equipped with a rod along the entire side which is utilized for attaching a compression band and a shoulder about one inch wide which rides over this rod is constructed. After the lead sheet is placed on the table, the patient is roentgenoscoped and the desired part brought into the field and the 11 × 14 cassette with strips of wood placed in the Bucky tray. The grid is moved so that the edge of the film corresponds to the overhanging portion of the indicator and then locked in place. After the first exposure, the grid is moved so that the fastening nut corresponds with the second notch, and for the third exposure moved to the third notch. After this the cassette is shifted 5½ inches in the tray and is in place for the fourth exposure, and others taken as above described. Multiple exposures can be taken with a considerable saving in time and expense. Two illustrations are given of multiple exposures of pylorus and duodenum, and also of the gall-bladder.

THOMS, HERBERT. X-ray pelvimetry—a simplified technique. *Surg., Gynec. & Obst.*, 1927, xlv, 827–828.

One of the chief essentials is that the patient shall be placed in such a position that the superior strait becomes parallel to the sensitive plate. This is possible when the patient is in a semirecumbent position with the back more or less arched.

In order to determine the level of this plane two points are identified on the external surface of the body; one on the anterior and upper border of the symphysis pubis and the other posteriorly at the depression just below the spine of the fourth lumbar vertebra. An imaginary line between these points traverses the anteroposterior diameter of the superior strait. For purposes of identification a small strip of adhesive is placed over the posterior landmark. By means of calipers these two points are placed equidistant from the plane upon which the patient sits. This distance is also measured for purposes to be explained later.

With the patient in position the tube is centered by means of a plumb-line and placed at a distance 36 in. above the sensitive plate over a point 5 cm. posterior to the upper and posterior border of the symphysis.

After the exposure is made the patient is removed from the apparatus with the tube and sensitive plate still in position. The distance of the height of the superior strait above the plane upon which the patient sat having previously been determined with calipers, a lead sheet is placed in the same plane and another flash exposure made on the same film. The lead sheet used is about 1 cm. thick and contains a row of very small holes exactly 1 cm. apart, every fifth hole being marked by a second offset hole.

On developing the plate an outline of the superior strait is readily seen slightly and equally enlarged, with a series of dots through the plane representing the "corrected" centimeters. From this the anteroposterior diameter may be read off directly with the aid of a pair of calipers. This procedure is regarded as one of unusual simplicity.

ULLMANN, K. Effect of roentgen rays on hair growth and hair pigment. *Strahlentherapie*, 1928, xxix, 291–302.

Literature is cited showing that there is sometimes an increased growth of hair with darker pigment following roentgen irradiation with low doses or the action of secondary radiation on parts of skin exposed to it unintentionally in treatment of the scalp and beard. Microscopic examination also shows greater development of pigment, especially in the medulla of the hair. This increased formation of pigment can also be demonstrated in the new hair that grows from the root after epilation. This effect of roentgen rays on hair has been overlooked as the rays have generally been used in large doses for purposes of epilation. It is probable that with weak irradiation and the selection of certain qualities of rays and particularly with finer methods of measurement the rays could be used cosmetically to bring about growth of hair. This is evidently a sort of rejuvenation of tissue due to stimulant action of the rays.

WALCH, H., DEN HOED, D., and KOOPMAN, L. J. Comparison of the absolute R unit with the French R unit. *Strahlentherapie*, 1928, xxix, 524–526.

Measurements are reported which show, as do the examinations made by other investigators, that Solomon's ionometer is not sufficiently independent of tension, and different instruments made by the same factory give different results. The authors think that this proves that this instrument cannot be used as a standard. But Solomon's method of measurement can be used for control anywhere where radium is available.

WALTER, B. On the best formulas for calculating the absorption of roentgen rays by any substance. II. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1927, xxxv, 1308–1310.

A more precise analysis has been made of the values given in the first communication covering the absorptions reported for homogeneous radiations. As a result, the Richtmyer formulas have been revised slightly to yield better correspondence with experimental observations. The improved Richtmyer equations are:

$$\frac{\mu'}{\rho} = 0.0160 \times \frac{N^{3.94}}{A} \times \lambda^3$$

$$\frac{\mu''}{\rho} = 0.000522 \times \frac{N^{4.30}}{A} \times \lambda^3$$

Recently Allen has proposed a new pair of equations closely resembling the Richtmyer type. These are:

$$\frac{\mu'}{\rho} = 0.0132 \frac{N^4}{A} \times \lambda^{2.92}$$

$$\frac{\mu''}{\rho} = 0.00181 \frac{N^4}{A} \times \lambda^{2.92}$$

The author presents a comparison of the individual and mean deviations from the true values of absorption coefficients for the elements carbon, aluminum, iron, copper, molybdenum, silver, tin, tungsten, platinum, gold and lead as calculated by the original Richtmyer equations, the author's modification of these equations and Allen's new equations.

A tabulation shows that the Allen formulas, although they yield fairly close agreement in the case of some elements, show for the series above enumerated an average deviation greater than that given by the other two formula pairs. The original Richtmyer formulas yield smaller average deviations but the smallest is given by the author's formula pair. This average deviation is 20 per cent less than that yielded by the Richtmyer formulas for absorption of roentgen rays of wave length between 0.1 and 1.0 Å units.

WALTER, B. The best formulas for the calculation of the absorption of roentgen rays by any substance. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1927, xxxv, 929–947.

As is well known, the absorption of roentgen rays by any substance can be calculated approximately from the atomic weights of its components by means of certain formulas. The equations tested by the author were the following:

(1) That of R. Glocker based on the old work of Barckla who employed radiations not entirely homogeneous. These formulas are as follows:

$$\frac{\mu'}{\rho} = 0.0195N^{2.58}\lambda^{2.8}$$

$$\frac{\mu''}{\rho} = 0.0004N^{3.14}\lambda^{2.8}$$

where μ is the absorption coefficient, ρ is the specific gravity of the substance, N is the atomic number and λ the wave length in Å units.

(2) The formulas of Allen which were obtained with somewhat more homogenous roentgen rays:

$$\frac{\mu'}{\rho} = 0.00782N^{2.92}\lambda^{2.92}$$

$$\frac{\mu''}{\rho} = 0.00100N^{2.92}\lambda^{2.92}$$

(3) The formulas of Richtmyer based on his extensive measurements of atomic absorption coefficients:

$$\frac{\mu'}{\rho} = 0.0136\frac{N^4}{A}\lambda^3$$

$$\frac{\mu''}{\rho} = 0.000529\frac{N^{4.3}}{A}\lambda^3$$

In this last equation A is the atomic weight of the element.

The author subjected these equations to test and found that the last, the equations of Richtmyer, gave the smallest deviations from experimentally determined values. Nevertheless, in special cases, errors of 20 to 30 per cent of the actual value are shown even by this pair of equations. In 80 to 90 per cent of the cases under examination the magnitude of deviation given by these equations was less than 10 per cent. The equations of Glocker yield deviations twice as great, while the deviations in the Allen equations lie between the two extremes.

From the mass absorption coefficients of the chemical elements, it is possible to calculate by means of the following equation

$$\frac{\mu}{\rho} \times m = \frac{\mu_1}{\rho_1} \times m_1 + \frac{\mu_2}{\rho_2} \times m_2 + \ldots$$

the absorptions of any chemical compound or any physical mixture of compounds provided

the proportions of the elements are known. In this last equation $\frac{\mu_1}{\rho_1}, \frac{\mu_2}{\rho_2}, \ldots$ represent the absorption coefficients of the various elements, and m_1, m_2 the corresponding masses.

WUCHERPFENNIG, V. Dosage with Sabouraud tablets based on the aluminum half-value layer. *Strablentherapie*, 1927, xxvii, 353–357.

The Sabouraud tablet is very much dependent on wave length and in order to determine dosage with it correctly in roentgen surface therapy the quality of the roentgen rays must be taken into consideration as is done in the biological gauging of Ritter, Rost and Krüger. But the quality of the rays is influenced by the height of the tension, the tension curve, the "range" of the roentgen tube and the thickness of the glass wall of the tube. The filter action of the glass wall of the tube may in some cases be equal to that of 0.5 mm. aluminum. The extent of all these sources of error is discussed and the aluminum half-value layer proposed as a measure of quality.

YAMASAKI, IWAO. Radium hemolysis. *Strablentherapie*, 1928, xxix, 363–366.

Pronounced hemolysis by radium emanation was observed by Salomonsen and Dreyer. The examinations were first made in hanging drops and then in Zeiss-Thoma counting chambers. By means of his blood agar plate method W. Hausmann demonstrated that the beta rays of radium also cause hemolysis and that in radium preparations of relatively low radium content these rays alone produce the effect. In experiments with very strong radium carriers and in radium preparations in which the radium was compressed within a very small space, Hausmann and Kerl found that with the use of suitable filters hemolysis was also brought about by gamma irradiation. Many authors formerly reported that hemolysis of red blood corpuscles could not be brought about by roentgen rays but Hausmann and Kerl, in view of the hemolytic action of gamma radium rays concluded that it was probable that hemolysis could also be brought about by roentgen rays and Holthusen has since proved this.

But in all these experiments the hemolysis was brought about in the presence of atmospheric oxygen; at the instigation of Hausmann the author undertook experiments to determine whether radium produces hemolysis in a nitrogen atmosphere. The experiments, the details of which are described, show that it does. In some of the experiments radium hemolysis was even stronger in the nitrogen matosphere than in ordinary air.

Subscribers to THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY *visiting New York City, are invited to make the office of* THE JOURNAL *(76 Fifth Avenue, New York) their headquarters. Mail, packages or bundles may be addressed in our care. Hotel reservations will gladly be made for those advising us in advance; kindly notify us in detail as to requirements and prices. List of operations in New York hospitals on file in our office daily.*